3

Exposure Assessment

This chapter describes the model used to estimate the occurrence of *Escherichia coli* O157:H7 in single servings of ground beef. This exposure model is divided into three modules: production, slaughter, and preparation. The production module estimates the occurrence of *E. coli* O157:H7 infection in two populations of live cattle: culled breeding cattle (cows and bulls) and cattle fed specifically for slaughter (steers and heifers). The slaughter module estimates the occurrence and extent of *E. coli* O157:H7 on carcasses and in beef trim combined in 2,000-pound combo bins or 60-pound boxes. The preparation module estimates the occurrence of *E. coli* O157:H7 in single servings of cooked ground beef. When appropriate, the effects of storage (e.g., chilling) and cooking are included throughout the model to account for organism growth or decline with resultant increased or decreased numbers of *E. coli* O157:H7. Exposure to *E. coli* O157:H7-contaminated ground beef servings was analyzed by age of the consumer and location where the meal was consumed (i.e., home or away from home). Each module of the exposure assessment model—production, slaughter, and preparation—yields one or more output distributions that serve either as inputs to the next module or as summary outputs.

PRODUCTION MODULE

The production module estimates the prevalence of *E. coli* O157:H7-infected cattle entering U.S. slaughter plants. It models culled breeding cattle (cows and bulls) and feedlot cattle (steers and heifers) at their points of origin through transit to the slaughter plant.

We know that *E. coli* O157:H7-infected cattle entering the slaughter process may influence the contamination of ground beef. A determination of the quantitative association between the incoming status of cattle and the outgoing status of harvested meat is critical in this exposure assessment. This quantitative correlation between pre-harvest and post-harvest contamination is best predicted using fecal *E. coli* O157:H7 prevalence data (Elder et al. 2000).

Explanation of Scope

The *E. coli* O157:H7 exposure assessment starts where beef production begins—at the farm. Most evidence on the occurrence and distribution of this organism in U.S. livestock was collected during surveys of farms and feedlots. Therefore, estimating the proportion of *E. coli* O157:H7-infected cattle at slaughter begins with estimating the proportion of infected cattle on the farm.

Imported beef is assumed to originate from countries whose *E. coli* O157:H7 epidemiology is similar to the United States. Approximately 15% of the fresh, chilled, and frozen beef and veal consumed in the United States is imported, and 90% of imports originate in Australia, New Zealand, and Canada (APHIS:VS:CEAH 1994). Specific data regarding the prevalence of *E. coli* O157:H7 in beef imported from various countries are lacking, and published surveillance data from the three major exporters to the U.S. are variable. However, evidence indicates that *E. coli* O157:H7 occurs in Australian, New Zealand, and Canadian cattle and humans (Robins–Browne et al. 1998; New Zealand Public Health Report 2000; Spika et al. 1998). In general, this evidence does not suggest that the prevalence of *E. coli* O157:H7 is dramatically greater in those countries than in the United States. Because this analysis intends to model all ground beef consumed in the United States, we assume that the share of imported ground beef that is contaminated is similar to the share of domestic ground beef that is contaminated.

The prevalence of infected cattle entering slaughter plants may be reduced through actions on the farm or feedlot. Many risk factors thought to influence *E. coli* O157:H7 status in cattle apply to whole herds rather than to individual cattle. For example, certain feed or feeding practices are hypothesized to elevate the probability of cattle becoming colonized with *E. coli* O157:H7 (Dargatz et al. 1997; Hancock et al. 1997b, 1998a; Herriot et al. 1998; Cray et al. 1998; Diez–Gonzales et al. 1998). Therefore, mitigation strategies typically target herd-level risk factors for *E. coli* O157:H7 control. For example, vaccination for *E. coli* O157:H7 would likely be applied at the herd level (Jordan et al. 1999; Gyles 1998).

Culled breeding cattle and feedlot cattle are separately modeled in this risk assessment. The slaughter, processing, and distribution of meat from these types of cattle are different. Furthermore, sampling evidence suggests that there may be differences in *E. coli* O157:H7 prevalence between these two types of cattle.

Breeding cattle comprise animals from dairy and beef cow-calf herds. In both types of breeding herds, mature cattle are bred to produce milk and calves. About 20% of all cattle slaughtered in the United States are breeding cattle (FSIS 1998). Feedlot cattle are steers and heifers sent to slaughter from feedlots. About 80% of all cattle slaughtered in the United States are feedlot cattle (FSIS 1998).

Definition of Key Terms

The following key terms are used throughout this module:

- Prevalence is the proportion of infected herds or individual cattle in a population.
- Herd prevalence is the proportion of herds with one or more *E. coli* O157:H7-infected cattle when the reference population is all herds of one type—for example, breeding herds.
- Apparent herd prevalence is the proportion of herds with one or more test-positive cattle detected among all herds sampled. Positive cattle are those animals that were diagnosed as infected or contaminated, based on testing. It is assumed that when microbiologic culture is used, all test-positive cattle are truly infected. “Infected” refers to cattle whose

intestinal tracts are colonized with the *E. coli* O157:H7 organism. "Contaminated" refers to cattle whose hides, hair, or hooves have some *E. coli* O157:H7 organisms residing on them. At present, no studies have specifically addressed the occurrence of contaminated cattle in herds, so the prevalence of infected herds is estimated based exclusively on infected cattle evidence. Given the limited understanding of the ecology of *E. coli* O157:H7 in cattle herds, it is assumed that contaminated cattle can only reside within herds that have one or more infected cattle.

- True herd prevalence is estimated by adjusting apparent herd prevalence observed in surveys with herd sensitivity.
- Herd sensitivity is the proportion of infected herds that, when tested, are detected as *E. coli* O157:H7-positive. Herd sensitivity is dependent on the number of samples collected within herds and the detectable prevalence of infected animals in the infected herds.
- Within-herd prevalence is the proportion of infected cattle when the reference population is the cattle within a specific infected herd. By convention, within-herd prevalence estimates only apply to infected herds. By definition, noninfected herds have a within-herd prevalence of 0%.
- Apparent within-herd prevalence is the proportion of *E. coli* O157:H7-positive cattle detected in a sample of cattle from an infected herd.
- True within-herd prevalence is estimated by adjusting the apparent within-herd prevalence observed in surveys by test sensitivity.
- Test sensitivity is the proportion of infected cattle that, when tested, are detected as *E. coli* O157:H7-positive using a particular diagnostic test. Test sensitivity is a complex parameter that incorporates variability in sample collection and handling and in the biological properties of the sample.

Production Module Segments

The production module comprises three segments: on-farm, transportation, and slaughter plant intake. As noted previously, culled breeding cattle ("breeding herds") are considered separately (Figure 3-1A) from feedlot cattle ("feedlots") (Figure 3-1B). The on-farm segment estimates the prevalence of *E. coli* O157:H7-infected herds (herd prevalence) and of *E. coli* O157:H7-infected cattle in infected herds (within-herd prevalence). Variability of within-herd prevalence among all infected herds—and by season of the year—is also estimated. The transportation segment considers the effect of transit time and commingling on the transmission and amplification of *E. coli* O157:H7 infections. The slaughter plant intake segment considers the effect of clustering cattle as they enter the slaughter plant. The following sections describe data and analysis for each of these segments.

On-Farm Segment

Breeding Herd Prevalence

Herd prevalence is the proportion of all breeding herds that contain one or more infected cattle. It is assumed that herd prevalence remains constant over time at a national level.

Hypothetically, herd prevalence might change across seasons or years. Seasonal changes in herd prevalence have been suggested (Garber et al. 1999), but these changes are most reasonably explained as the result of seasonal changes in the within-herd prevalence for infected herds. Seasonal variation in within-herd prevalence has been previously reported (Hancock et al. 1994,

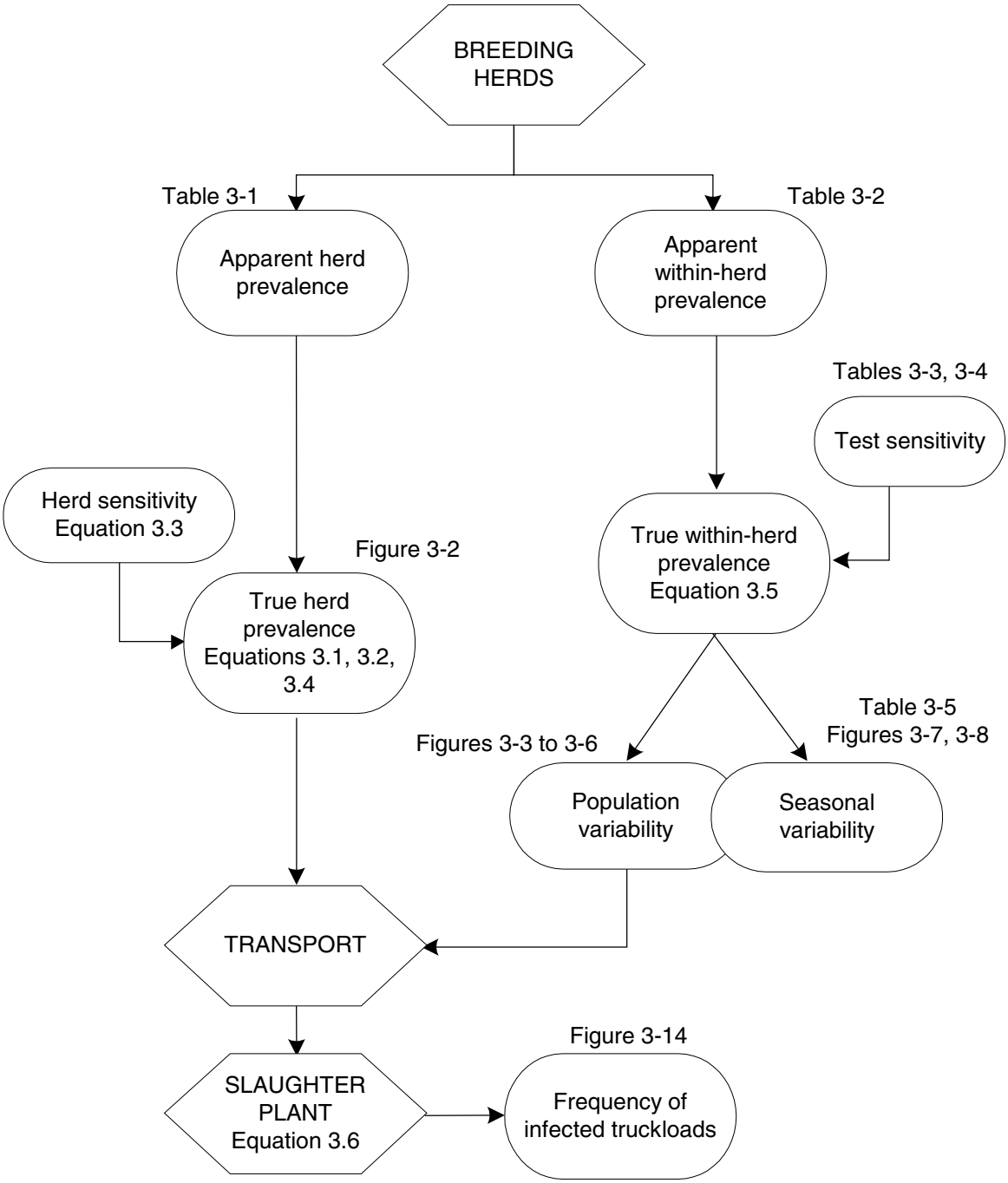

FIGURE 3-1A Production module flowchart for estimation of key variables for breeding herds (cows and bulls).

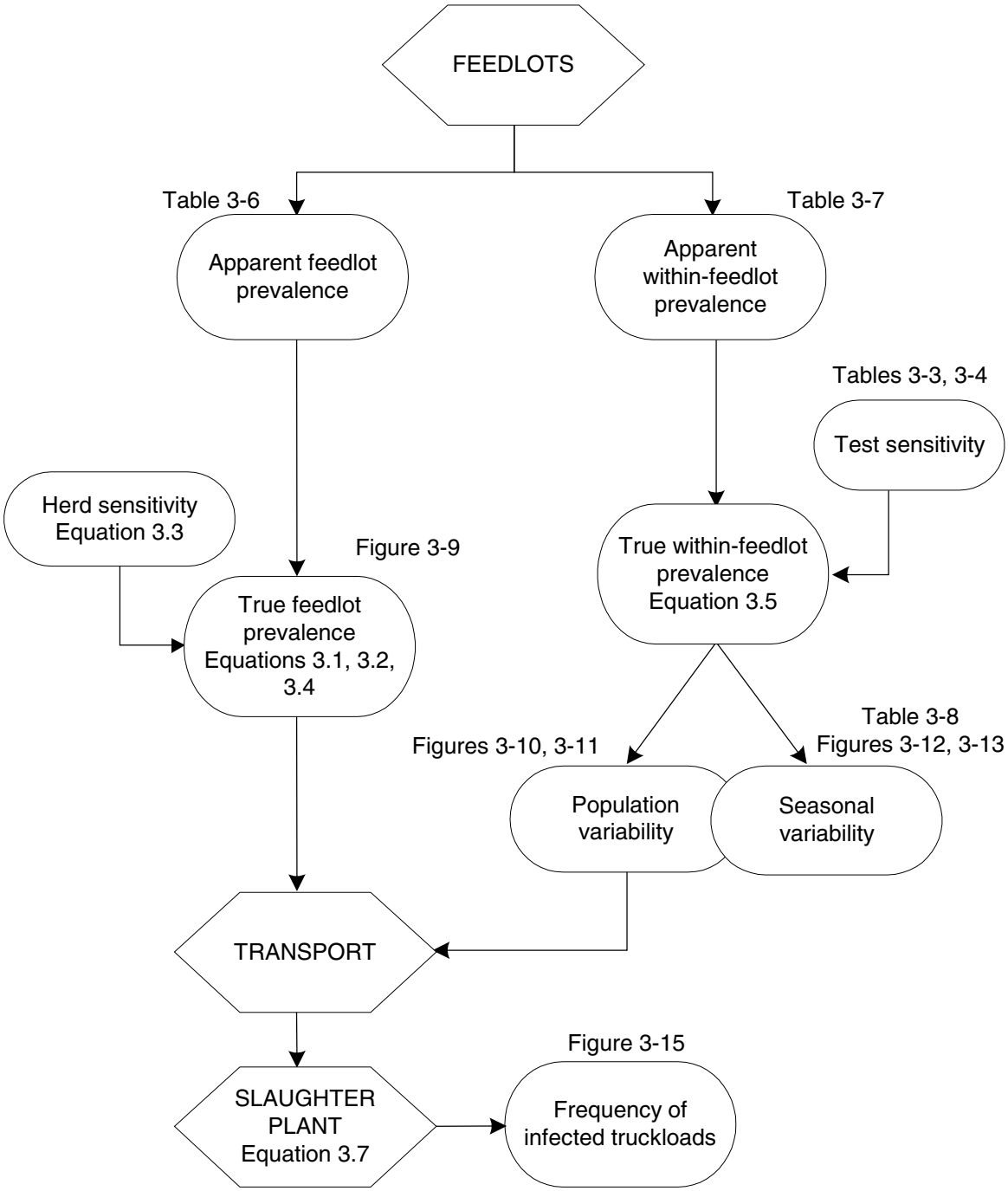

FIGURE 3-1B Production module flowchart for estimation of key variables for feedlot herds (steers and heifers).

1997b; Heuvelink et al. 1998). If within-herd prevalence varies by season, then the apparent herd prevalence detected in surveys will also vary in a similar pattern (assuming sample size within herds is constant). While herd prevalence might change across years, there is no empirical evidence supporting such a change in the past 5 years.

Herd prevalence is estimated using evidence that may have been generated from sampling herd subpopulations other than mature cattle. Yet evidence about the existence of *E. coli* O157:H7 within any age of cattle in a herd indicates that cows or bulls culled from that herd might be infected.

Apparent Breeding Herd Prevalence

Seven studies provide evidence regarding the apparent prevalence of infected breeding herds (Table 3-1). Nearly all studies sampled herds from multiple states in the United States.

TABLE 3-1 Evidence Used to Estimate Breeding Herd Prevalence

Study	Herds Tested	Positive Herds	Apparent Herd Prevalence	Average Samples Per Herd	Apparent Within-Herd Prevalence	Lab Methods	Months Sampled
Hancock et al. 1997a	13	9	69%	791	1.3%	0.1 g, SMACct	June–May
Hancock et al. 1997b	36	27	75%	360	1.8%	0.1 g, SMACct	July–December
Hancock et al. 1998a	6	6	100%	183	2.3%	0.1 g, SMACct	July–November
Garber et al. 1999	91	22	24%	58	4.0%	1 g, SMACct, TSB	February–July
Lagreid et al. 1999	15	13	87%	60	8.0%	10 g, IMS	October–November
Sargeant et al. 2000	10	10	100%	235	1.2%	10 g, IMS	January–December
Hancock 2001	20	18	90%	317	0.7%	0.1 g, SMACct	December–March, June–September

Note: g = grams of feces analyzed,
SMACct = sorbitol MacConkey media with cefixime and tellurite,
TSB = trypticase soy broth, and
IMS = immunomagnetic separation.

National studies have not shown any geographic clustering of *E. coli* O157:H7 among breeding herds (Garber et al. 1995, 1999). Therefore, U.S. herd prevalence data are pooled without regard for the region where the data were collected.

Hancock et al. (1997a) sampled 13 dairy herds in three northwestern states monthly for 1 year (1993 to 1994); 9 (69%) herds tested positive. Approximately 60 samples were collected on each visit from a combination of weaned heifers and adult cows. Apparent within-herd prevalence in the nine positive herds was 1%.

Hancock et al. (1997b) sampled 36 dairy herds in three northwestern states from July to December 1994; 27 (75%) of the 36 tested herds were positive. In each herd, 60 fecal samples

from post-weaned heifers were collected once a month, and about 2% of cattle within infected herds were positive.

Hancock et al. (1998a) also sampled six dairy herds in three northwestern states from July to November 1996. In each herd, 60 fecal samples from post-weaned heifers were collected once a month for 3 months. All herds tested positive. Apparent within-herd prevalence was 2.3%.

Garber et al. (1999) report on a national survey of the U.S. dairy industry conducted by the U.S. Department of Agriculture (USDA) from February to July 1996. Fecal samples were collected from 91 dairy herds across the United States, and 22 herds were found to have one or more test-positive cattle. Within each herd, the average number of samples collected was 58, and about 4% of sampled cattle in the positive herds were found to be *E. coli* O157:H7-positive.

Lagreid et al. (1999) sampled 15 cow-calf herds across five midwestern states in October and November 1997; 13 (87%) herds tested positive. In each herd, 60 fecal samples from weaned calves were collected. This study used more sensitive lab methods than many studies that preceded it. Therefore, the apparent within-herd prevalence (8%) found in this study reflects the improved capacity of that test to detect positive cattle.

Sargeant et al. (2000) sampled 10 Kansas cow-calf herds once a month for 1 year (1996 to 1997), and all 10 herds tested positive. On each visit, about 10% of the cow and bull herd was sampled (~20 head per month). This study also used very sensitive test methods but found an apparent within-herd prevalence (~1%) more consistent with studies using less sensitive methods.

Hancock (2001) is completing a study of 30 dairy herds in two northwestern states. Twenty of these herds have been sampled during the winter (December through March) and summer (June through September). Eighteen of these herds were found to contain at least one positive cattle. Apparent within-herd prevalence for adult cattle is 0.7% using moderately sensitive test methods.

True Breeding Herd Prevalence

True herd prevalence is estimated from apparent herd prevalence using Bayes Theorem:

$$f(\Phi \mid y) = \frac{f(y \mid \Phi)\, f(\Phi)}{\int_0^1 f(y \mid \Phi)\, f(\Phi)\, d\Phi} \tag{3.1}$$

Equation 3.1 predicts the distribution for true herd prevalence (Φ), given apparent prevalence evidence (y). The function, $f(y \mid \Phi)$, is the likelihood of observing a particular sampling result (e.g., 27 positive herds in 36 sampled herds from Hancock et al. 1997b), given true herd prevalence Φ. This likelihood function depends on the herd sensitivity (HSens), the number of herds sampled in a study (N), and the number found positive (S):

$$f(y \mid \Phi) = \binom{N}{S} (\text{HSens} \times \Phi)^S \, (1 - \text{HSens} \times \Phi)^{N-S} \tag{3.2}$$

The herd sensitivity (HSens) of a particular survey was defined as

$$\text{HSens} = 1 - \int (1 - p_i)^n \, f(p_i)\, dp, \tag{3.3}$$

where p_i is the apparent within-herd prevalence in herd i, $f(p_i)$ is the frequency of p_i, and n is the number of samples collected in each herd.

Using Monte Carlo methods, HSens was estimated to be 0.75 for Garber et al. (1999), 0.86 for Lagreid et al. (1999) and Sargeant et al. (2000), 0.89 for Hancock et al. (1998a) and Hancock (2001), 0.96 for Hancock et al. (1997b), and 0.99 for Hancock et al. (1997a). Apparent within-herd prevalence was assumed to be an exponential distribution (as discussed in the "Within-Breeding Herd Prevalence" section). Average within-herd prevalence was modeled using a beta(s+1,n–s+1) distribution, where *s* was the number of test-positive cattle in detected herds and *n* was the total cattle tested in detected herds (Vose 1996).

True breeding herd prevalence (Figure 3-2) was estimated by combining the results from Equation 3.2 across all seven studies using Equation 3.4:

$$f(\theta \mid x_i) = \frac{f(x_i \mid \theta)\, f(\theta_{i-1})}{\int_0^1 f(x_i \mid \theta)\, f(\theta_{i-1})\, d\theta} \tag{3.4}$$

where x_i reflects the evidence provided by study *i*, and $f(\theta_{i-1})$ is the prior distribution for breeding herd prevalence based on evidence provided by study *i–1*.

Figure 3-2 suggests that breeding herd prevalence is most likely 65%, but it could be as low as 50% or as high as 80% based on the available evidence. Therefore, the majority of breeding herds in the United States are predicted to contain one or more *E. coli* O157:H7-infected cattle.

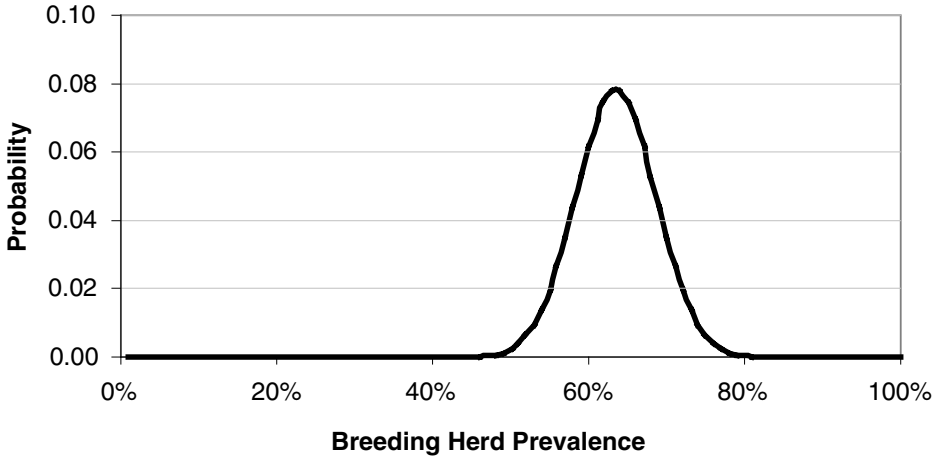

FIGURE 3-2 Resultant uncertainty distribution for true breeding herd prevalence after analysis of data in Table 3-1.

As defined in this risk assessment, breeding herds comprise dairy and cow-calf herds. Although most evidence on breeding herds was collected in dairy herds, two studies exclusively sampled cattle in cow-calf herds (Lagreid et al. 1999; Sargeant et al. 2000). Dairy cows are usually managed intensively. They are gathered at least twice daily and often confined to lots or pastures where contact between individuals is likely to occur. Commercial dairies are also very busy operations: milk trucks, feed delivery vehicles, and other visitors are common. Cows in cow-calf herds are less intensively managed. These cows usually live on large pastures throughout the year. Hypothetically, the potential for fecal-oral spread of *E. coli* O157:H7 is greater for dairy herds than for beef herds based on these management differences. Furthermore, the potential for introduction of *E. coli* O157:H7 into a dairy would seemingly be greater given the increased traffic and congestion in such operations. Yet the studies show that cow-calf herds

are no less likely to be infected than dairy herds (i.e., Lagreid et al. [1999] found 87%—and Sargeant et al. [2000] found 100%—of cow-calf herds they studied positive). Although the evidence is limited, it suggests that dairy and cow-calf herds are similar with respect to *E. coli* O157:H7.

Within-Breeding Herd Prevalence

Within-herd prevalence is the proportion of infected cattle that an infected herd might send to slaughter. Culled breeding cattle sent to slaughter are a subset of these herds. Within-herd prevalence in this model applies to just these cattle.

Apparent Within-Breeding Herd Prevalence

Within-herd prevalence varies among the population of all infected herds. If all the infected herds could be examined at a given point in time, differences in within-herd prevalence among these herds could be observed. Within-herd prevalence also varies systematically among infected herds by season (Hancock et al. 2001). Therefore, within-herd prevalence is modeled as a frequency distribution to reflect population variability, but the frequency distribution is adjusted to reflect seasonal patterns.

Population variability. Two studies provide evidence about the population variability of within-herd prevalence among known-infected herds (Hancock et al. 1997b; Garber et al. 1999). Both studies included sufficient herds (i.e., 27 and 22 herds) and samples to estimate a distribution.

Figure 3-3 is a histogram of within-herd prevalence from a study that sampled dairy heifers in three northwestern states between July and December 1994 (Hancock et al. 1997b). This histogram suggests a declining frequency of herds as within-herd prevalence increases. Its mean and standard deviation are 1.9% and 1.3%, respectively. Hypothetically, such a histogram might be generated from an exponential distribution.

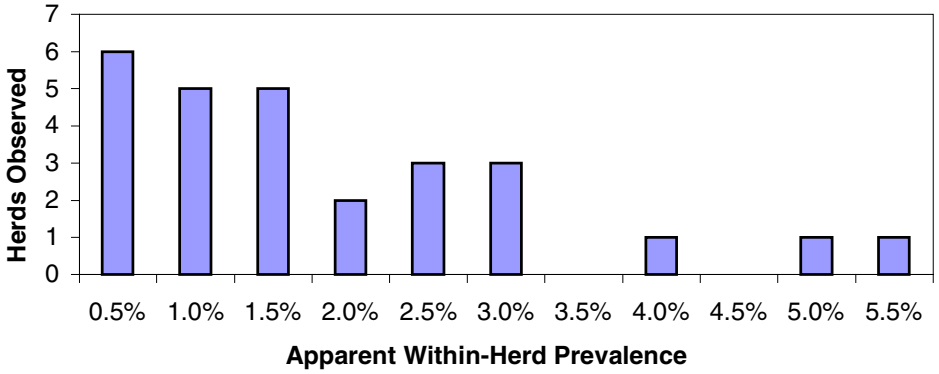

FIGURE 3-3 Evidence on the distribution of within-herd prevalence of *E. coli* O157:H7 among 27 infected herds (adapted from Hancock et al. 1997b).

The exponential distribution has one parameter, β, that is both its mean and standard deviation. A comparison of the Hancock et al. (1997b) data to predictions from an exponential distribution with $\beta = 1.9\%$ shows general agreement (Figure 3-4). Using a Chi-square statistic, the hypothesis that the observed and expected results were equivalent was not rejected ($\chi^2 = 0.92$, $p>0.05$). Degrees of freedom for this test were determined using Scott's normal approximation (Vose 1996).

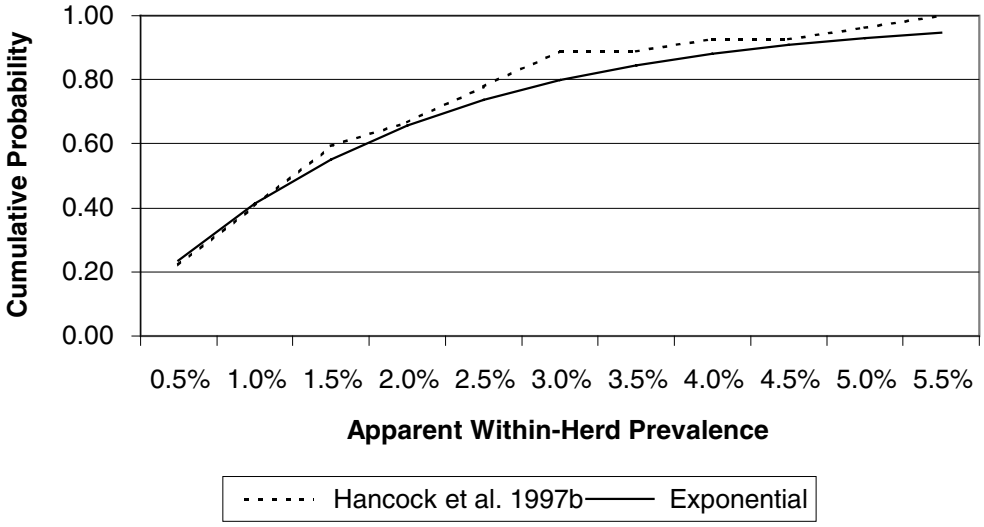

FIGURE 3-4 Comparison of observed and expected cumulative probabilities for within-herd prevalence of *E. coli* O157:H7.

Figure 3-5 is a histogram of within-herd prevalence from a national USDA survey of dairy cows (Garber et al. 1999). These data also reasonably fit an exponential distribution ($\chi^2 = 9.2$, $p>0.05$) (Figure 3-6).

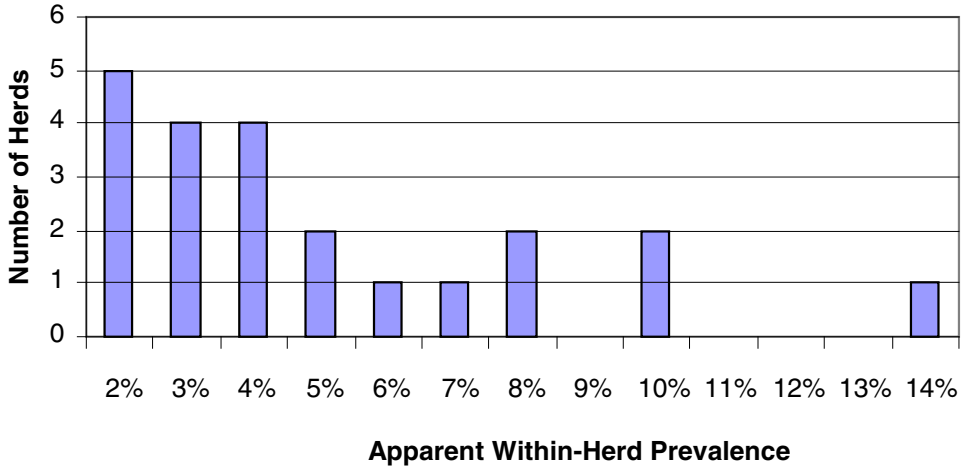

FIGURE 3-5 Evidence on the distribution of within-herd prevalence of *E. coli* O157:H7 among 22 infected herds (adapted from Garber et al. 1999).

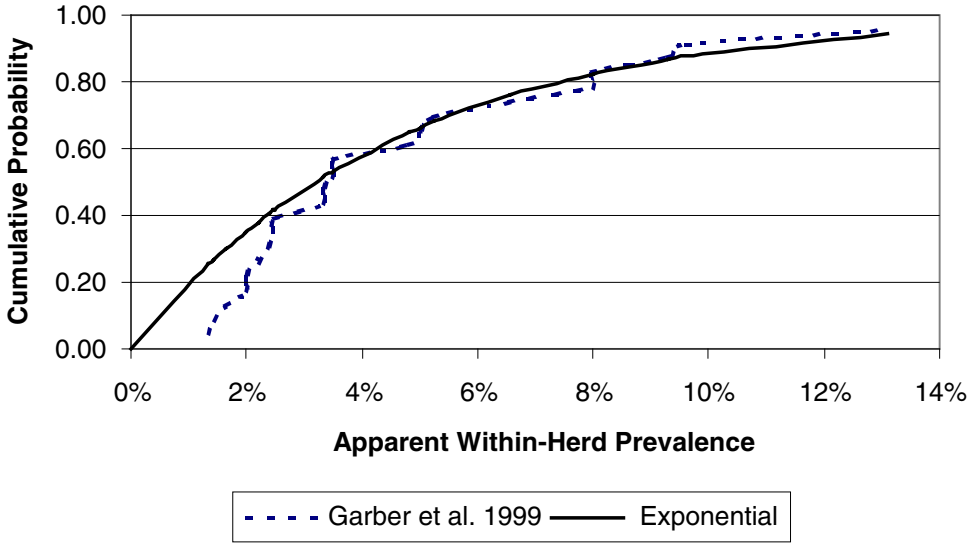

FIGURE 3-6 Comparison of observed and expected cumulative probabilities for within-herd prevalence of *E. coli* O157:H7.

Other prevalence studies either sampled very few infected herds (e.g., Besser et al. 1997; Hancock et al. 1994; Sargeant et al. 2000) or did not collect many samples within each infected herd (Rice et al. 1997). A histogram of within-herd prevalence generated from these studies would not adequately depict its variability. Yet by assuming that within-herd prevalence of *E. coli* O157:H7 fits an exponential distribution, the results from these studies can be used to estimate the average within-herd prevalence. The exponential distribution then describes the variability of within-herd prevalence based on this average.

Seasonal variability. Evidence of a summer peak in cattle *E. coli* O157:H7 prevalence (Hancock et al. 1994; Garber et al. 1999; Hancock et al. 1997a; Heuvelink et al. 1998; Van Donkersgoed et al. 1999) suggests that the greatest *E. coli* O157:H7 prevalence occurs between June and September. It is thought that a summer rise in prevalence results from on-farm environmental conditions that provoke increased transmission of *E. coli* O157:H7 among cattle (Hancock 2001). For example, if feed and water are important in the transmission of *E. coli* O157:H7 to cattle within a herd, then summer ambient temperatures might induce substantial growth of *E. coli* O157:H7 in the feed and water that cattle ingest and result in more infected cattle.

One study of cattle in Canada found at least a fourfold difference in *E. coli* O157:H7 fecal prevalence between samples collected in the winter and summer (Van Donkersgoed et al. 1999). The greatest fecal prevalence was observed between June and August. In a national study of U.S. dairies, herds sampled between May and July were nearly eight times more likely to be fecal positive than those sampled between February and April (Garber et al. 1999). Longitudinal studies that followed the same infected herds for a full year have found a three- to sixfold difference in prevalence between winter and summer (Hancock et al. 1997a; Heuvelink et al. 1998). Nevertheless, a yearlong study of 10 cow-calf herds did not demonstrate any seasonal difference in prevalence (Sargeant et al. 2000).

To model the effect of season, within-herd prevalence is estimated for two periods: June to September, which constitutes the high prevalence season, and the other months of the year, which constitute the low prevalence season. Each season's average within-herd prevalence is

estimated. During each season, population variability of within-herd prevalence is modeled via the exponential distribution.

Evidence of Apparent Within-Breeding Herd Prevalence

Six studies provide evidence on apparent within-herd prevalence of infected adult cattle in U.S. breeding herds (Table 3-2). Although all of these studies sampled adult cows and bulls, the study design, sampling scheme, and culturing methods often differed.

TABLE 3-2 Evidence Used to Estimate Within-Herd Prevalence of *E. coli* O157:H7 in Breeding Herds

Study	Number Tested in Positive Herds	Positive in Positive Herds	Apparent Within-Herd Prevalence	Lab Methods	Months Sampled
Hancock et al. 1994	458	20	4.4%	0.1 g, SMAC	June–July, September
Besser et al. 1997	2074	53	2.6%	0.1 g, SMACct	January–December
Rice et al. 1997	75	7	9.3%	0.1 g, SMACct	July–December
Garber et al. 1999	1268	51	4.1%	1 g, SMACct, TSB	February–July
Sargeant et al. 2000	2348	29	1.2%	10 g, IMS	January–December
Hancock et al. 2001	5709	38	0.7%	0.1 g, SMACct	December–March, June–September

Note: g = grams of feces analyzed,
SMAC = sorbitol MacConkey media,
SMACct = sorbitol MacConkey media with cefixime and tellurite,
TSB = trypticase soy broth, and
IMS = immunomagnetic separation.

Hancock et al. (1994) surveyed 25 cow-calf herds in Washington, and 4 (16%) were positive. Within those positive herds, about 4% of cows were fecal positive for *E. coli* O157:H7. Sampling was conducted in June, July, and September 1992.

Besser et al. (1997) conducted a yearlong study of 10 dairy herds in Washington, and 4 (40%) were positive. Within those positive herds, the prevalence of positive cattle was about 3%. Sampling was completed during 1993 and 1994.

Rice et al. (1997) sampled cows culled from 13 positive dairy herds in Idaho, Oregon, and Washington. This study found 9% of cattle from positive herds to be fecal positive. Sampling was conducted between July and December 1994.

In Garber et al. (1999), 22 infected dairy herds were detected as part of a national USDA survey. Four percent of the cows sampled in the positive herds were *E. coli* O157:H7-positive. Sampling was conducted between February and July 1996.

Sargeant et al. (2000) detected 10 positive Kansas cow-calf herds in a yearlong study. About 1% of the cows were fecal positive. The study was conducted between December 1996 and December 1997.

Hancock et al. (2001) are completing a study in which 18 positive herds have been detected. Almost 1% of cattle sampled in positive herds are positive. These results reflect sampling conducted during 2000 and 2001.

True within-herd prevalence can be estimated from apparent within-herd prevalence (Martin et al. 1987):

$$\text{True Prevalence} = \frac{\text{Apparent Prevalence}}{\text{Test Sensitivity}} \tag{3.5}$$

Apparent prevalence is estimated as a beta(s+1,n–s+1), where s is the number of test positive cattle in a study and n is the total cattle tested in positive herds (Vose 1996). Test sensitivity is estimated from research evidence.

Test Sensitivity

The probability of observing a positive biological test result depends on test sensitivity. Both the culture methods used and the quantity of sample collected affect test sensitivity. The absolute sensitivity of microbiological tests applied to naturally-infected cattle has not been established because there is no suitable "gold" standard for determining the true infection status of cattle. Nevertheless, Sanderson et al. (1995) have evaluated the sensitivity of culturing methods using 24 naturally-infected dairy cattle (Table 3-3). These relative sensitivity measures included the effects of different culture methods and sample quantities. The least sensitive method had a relative sensitivity of 0.33—in other words, only 33% of the infected cattle were found positive using this method. The most sensitive method had a relative sensitivity of 0.79.

TABLE 3-3 Relative Test Sensitivity of Lab Methods. Twenty-four test-positive cattle were detected using different sample quantities (0.1 gram and 10 grams) and plating media.

Lab Methods	Number Positive	Relative Sensitivity
0.1 gram, TSBcv, SMACc	8	0.33
0.1 gram, TSBcv, SMACct	14	0.58
10 gram, TSBcv, SMACct	19	0.79
Total positives	24	

Note: TSBcv = trypticase soy broth with cefixime and vancomycin,
SMACc = sorbitol MacConkey media with cefixime, and
SMACct = sorbitol MacConkey media with cefixime and tellurite.

Source: Adapted from Sanderson et al. 1995.

The quantity of feces sampled from cattle influences test sensitivity because infected cattle shed *E. coli* O157:H7 in varying concentrations. Variability in *E. coli* O157:H7 fecal concentration from naturally-infected cattle has been reported (Zhao et al. 1995; Cassin et al. 1998). The range of feasible concentrations should extend to 10^7 to account for shedding levels infrequently observed in experimentally-infected adult cattle (Cray and Moon 1995). A minimum shedding concentration of 10^{-1} colony-forming units (CFU) per gram of feces can be assumed, based on a 10-gram sample. Plausible frequencies for this range of fecal concentrations are listed in Table 3-4.

TABLE 3-4 Calculation of the Probability of Detecting One or More Organisms Given the Sample Quantity, Concentration of Organisms per Gram of Feces, and Frequency (f[x]). Lambda (λ) equals the CFU per gram multiplied by the sample size. The sum of each column is the expected frequency of samples containing no *E. coli* O157:H7 organisms from a cross-section of infected cattle.

		P(x=0|λ)*f(x)		
CFU per Gram of Feces	f(x)	0.1 Gram Sample	1 Gram Sample	10 Gram Sample
0.1	0.12	0.117	0.107	0.043
1	0.12	0.107	0.043	0.000
10	0.12	0.043	0.000	0.000
100	0.12	0.000	0.000	0.000
1,000	0.06	0.000	0.000	0.000
10,000	0.35	0.000	0.000	0.000
100,000	0.09	0.000	0.000	0.000
1,000,000	0.02	0.000	0.000	0.000
10,000,000	0.01	0.000	0.000	0.000
Sum	1	0.267	0.150	0.043
1–Sum		0.733	0.850	0.957

Fecal prevalence studies have included 0.1-gram, 1-gram, and 10-gram sample quantities. The probability that a given sample quantity will not contain any organisms is predicted by the Poisson distribution, e^{-xz}, where x is concentration per gram of feces and z is the sample quantity in grams. If x is a distribution, then this probability is the expected value across all concentrations (i.e., $\Sigma f(x) \times e^{-xz}$), where f(x) is the frequency of concentration x.

The probability of a sample containing one or more organisms is equal to one minus the probability it contains no organisms. The probability that a sample size of 0.1, 1.0, and 10 grams will contain at least one organism is 0.73, 0.85, and 0.96, respectively (Table 3-4). Therefore, increasing the sample quantity from 0.1 grams to 10 grams results in 23% (= 96% – 73%) more samples with 1 or more *E. coli* O157:H7 organisms from infected cattle. Interestingly, when 0.1- and 10-gram samples were evaluated using the same enrichment and plating system (i.e., TSBcv, SMACct), the 10-gram sample detected 79% of infected cattle while the 0.1-gram sample detected 58% of these cattle, a difference of 21% (Table 3-3). Therefore, the observed difference in sensitivity between these methods approximates the effect of different sample quantities.

The test sensitivity applicable to the Besser et al. (1997), Rice et al. (1997), and Hancock et al. (2001) studies is shown in Table 3-3 (i.e., 0.58). The other within-herd prevalence studies used alternative methods for which test sensitivity is not directly reported.

The Garber et al. (1999) study used 1.0-gram samples and TSB-SMACct. Neither the TSB enrichment nor the 1.0-gram sample size is available from the results in Table 3-3. The TSBcv-SMACct culturing protocol detected 80% of samples experimentally spiked with *E. coli* O157:H7 (Sanderson et al. 1995). Yet a 1.0-gram sample from infected cattle is only 85% likely to contain *E. coli* O157:H7. Therefore, a 1.0-gram, TSBcv-SMACct protocol is predicted to detect 68% (85% × 80%) of infected cattle. In another experiment, the difference between the

TSB enrichment system and the TSBcv system equaled –10% (Sanderson et al. 1995). Therefore, the sensitivity for the 1.0-gram TSB-SMACct sampling protocol is estimated as 58%.

Hancock et al. (1994) used 0.1-gram samples and TSBv-SMAC. The SMAC plating system only detected 3% of samples experimentally spiked with *E. coli* O157:H7 (Sanderson et al. 1995). A 0.1-gram sample from infected cattle is only 73% likely to contain *E. coli* O157:H7. Therefore, the sensitivity of the 0.1 gram-TSBv-SMAC sampling protocol was estimated as 2% ($73\% \times 3\%$).

Sargeant et al. (2000) used 10-gram samples and immunomagnetic separation (IMS) with microbiologic culture to improve the detection of *E. coli* O157:H7 in fecal samples. The IMS process was found to have a sensitivity that was 20% greater than a single dilution microbiologic culture system (Sanderson et al. 1995). Therefore, sensitivity of the 10-gram IMS sampling protocol was estimated as 100%.

Test sensitivities in Table 3-3 and those generated above were used in Equation 3.5 to estimate true prevalence. Uncertainty regarding test sensitivity was modeled using beta distributions (Vose 1996).

True Within-Breeding Herd Prevalence

Seasonal variability. Examining the monthly prevalence evidence, there appears to be a high prevalence season (June to September) and a low prevalence season (October to May).

Three studies (Garber et al. 1999; Hancock et al. 1994, 2001) provide different sampling evidence for different months of the study. For example, Garber et al. (1999) sampled cattle from February through July. These data show that 7 of 193 cattle sampled in infected herds were fecal positive during the period from February to May. In contrast, 44 of 1,075 cattle sampled in infected herds during June and July were fecal positive.

Data collected for each month of the year were pooled. Prior to pooling, true within-herd prevalence for each study was estimated. Average within-herd prevalence was calculated for each month across all the applicable studies by weighting each study by the average cattle sampled per month in the study. Within-herd prevalence estimated for June to September was averaged to calculate within-herd prevalence during the high prevalence season. Similarly, within-herd prevalence during the low prevalence season was the average across October to May.

Table 3-5 illustrates this method of estimating seasonal averages using point estimates. Recall that true prevalence is a random variable estimated from two beta-distributed variables (apparent prevalence and test sensitivity). These point estimates illustrate one scenario when the averages of apparent prevalence and test sensitivity are used. To calculate true averages, Monte Carlo methods were used to simulate the underlying distributions (Haas et al. 1999).

Figure 3-7 overlays a centered 3-month moving average curve upon nine illustrative iterations of the Monte Carlo model. The moving average curve is calculated from 1,000 iterations of the model and demonstrates a seasonal pattern of within-herd prevalence. Nevertheless, the limited data and estimation method also result in considerable uncertainty about the true monthly within-herd prevalence. A given month's estimate may be substantially influenced by the amount of available data (e.g., August) as well as the uncertainty in apparent prevalence and test sensitivity. Nevertheless, the volatility implied by the single iteration curves is dampened because the model only considers estimates of the high and low prevalence seasons.

Figure 3-8 shows the uncertainty about the seasonal averages. Despite the apparent overlap of the two seasonal distributions, there were 913 of 1,000 iterations of the Monte Carlo model in which the prevalence for June to September (high prevalence season) was greater than that for

TABLE 3-5 Point Estimates for Monthly True Within-Herd Prevalence for Each of Six Studies (Table 3-2). A weighted average for each month was calculated (based on average numbers of samples collected per month per study), and a seasonal average was calculated for the high and low prevalence seasons.

	Weighted Average						
Month	Hancock et al. 1994	Besser et al. 1997	Rice et al. 1997	Garber et al. 1999	Sargeant et al. 2000	Hancock et al. 2001	Average
January		4.5%			1.3%	1.0%	1.6%
February		4.5%		7.1%	1.3%	1.0%	2.6%
March		4.5%		7.1%	1.3%	1.0%	2.6%
April		4.5%		7.1%	1.3%		4.6%
May		4.5%		7.2%	1.3%		4.6%
June	45.3%	4.5%		7.2%	1.3%	1.4%	4.2%
July	66.8%	4.5%	18.0%	7.2%	1.3%	1.4%	5.0%
August		4.5%	18.0%		1.3%	1.4%	2.1%
September	75.6%	4.5%	18.0%		1.3%	1.4%	4.8%
October		4.5%	18.0%		1.3%		3.3%
November		4.5%	18.0%		1.3%		3.3%
December		4.5%	18.0%		1.3%	1.0%	1.7%
Weights	46	173	13	254	196	794	
October–May average		(low prevalence season)			3.0%		
June–September average		(high prevalence season)			4.0%		
January–December average					3.4%		

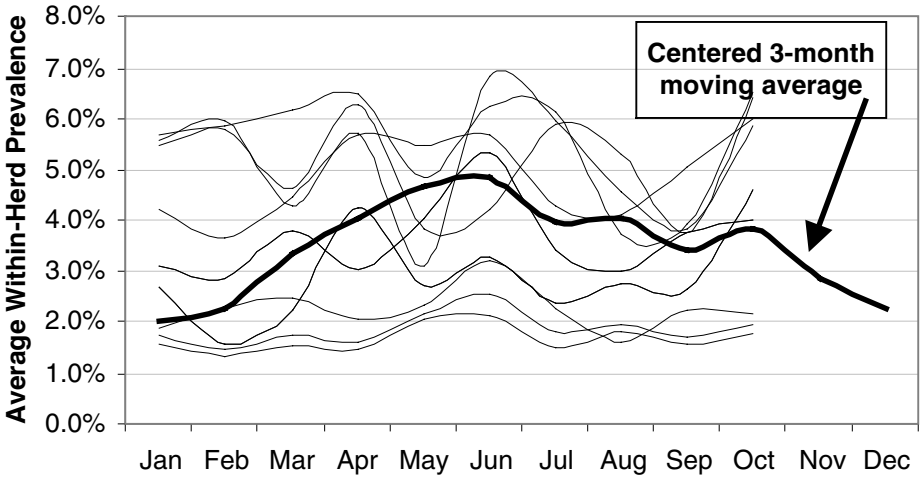

FIGURE 3-7 Estimated average monthly within-herd prevalence. This illustrated seasonal trend is based on 1,000 iterations of the model.

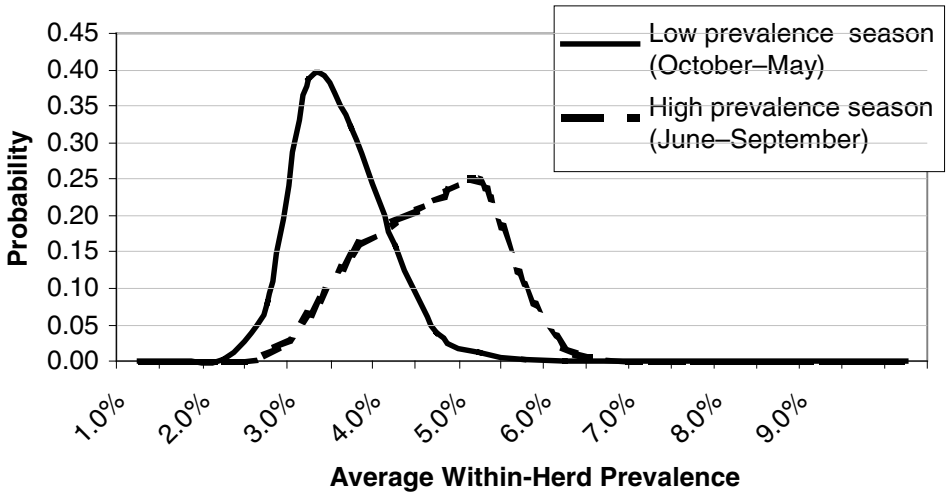

FIGURE 3-8 Uncertainty about low and high prevalence seasons' estimated average within-herd prevalence. These distributions were estimated using 1,000 Monte Carlo iterations of the model.

the rest of the year (low prevalence season). The averages of the low and high prevalence season distributions were 3.1% and 4.2%, respectively. Therefore, this analysis suggests that within-herd prevalence is increased 33% during June to September relative to the rest of the year. For comparison, the Sargeant et al. (2000) study found no evidence of change by season, and the Hancock et al. (2001) study found a 66% increase during June to September. These studies sampled adult cows during both the low and high prevalence seasons.

These results imply that prevalence within infected breeding herds during June to September varies around a greater average than during other months of the year. Consequently, cattle shipped to slaughter from infected herds during June to September are more likely to be infected than at other times. If cattle slaughtered during June to September are more likely to be infected, then the risk associated with ground beef produced from these cattle may also be elevated relative to other times of the year.

Feedlot Prevalence

As with breeding herds, the prevalence of infected feedlots is also assumed to be constant across time. The occurrence of *E. coli* O157:H7 in feedlots does not show any geographic clustering (Hancock et al. 1998b, 2001). Therefore, U.S. feedlot prevalence data are also pooled without regard for the region where the data were collected.

Apparent Feedlot Prevalence

Four studies provide evidence regarding the apparent prevalence of infected feedlots (Table 3-6). Feedlots sampled in each study came from multiple states.

Dargatz et al. (1997) report on a national survey conducted by USDA in 1994 (Hancock et al. 1997c). In this study, 100 feedlots were randomly selected throughout the United States; 63 feedlots were found to contain one or more positive cattle. Thirty fecal samples per pen were collected from four pens in each feedlot. About 3% of cattle sampled in positive feedlots were fecal positive.

TABLE 3-6 Evidence Used to Estimate Feedlot Prevalence

Study	Feedlots Tested	Positive Feedlots	Apparent Feedlot Prevalence	Average Samples per Feedlot	Apparent Within-Feedlot Prevalence	Lab Methods	Months Sampled
Dargatz et al. 1997	100	63	63%	120	3%	0.1 g, SMACct	October–December
Hancock et al. 1998b	6	6	100%	174	4%	0.1 g, SMACct	July–November
Smith 1999	5	5	100%	611	23%	10 g, IMS	June–September
Elder et al. 2000	29	21	72%	12	36%	10 g, IMS	July–August

Note: g = grams of feces analyzed,
SMACct = sorbitol MacConkey media with cefixime and tellurite, and
IMS = immunomagnetic separation.

Hancock et al. (1998b) completed a survey of six feedlots in Idaho, Oregon, and Washington during 1996. At least one positive cattle was detected in each feedlot. An average of 174 samples were collected per feedlot, and about 4% of cattle in positive feedlots were positive.

Smith (1999) sampled five midwestern feedlots, and all were found to contain positive cattle. Four to five pens were intensively sampled in each feedlot during a 3-month period during summer 1999. An average of 611 samples were collected per feedlot, and 23% of cattle in these feedlots were positive. This study used much more sensitive test methods than the previous studies.

Elder et al. (2000) also used very sensitive test methods to sample cattle at four midwestern slaughter plants in 1999. It was assumed that each lot of cattle sampled in this study represented a pen of cattle originating from a randomly selected feedlot. Of the 29 lots sampled, 21 were detected to contain one or more positive cattle. While an average of only 12 samples were collected per lot, 36% of the cattle were *E. coli* O157:H7-positive in positive lots.

True Feedlot Prevalence

To estimate true feedlot prevalence, the same methods were used as described for breeding herd prevalence (Equations 3.1 to 3.4). Herd sensitivity (HSens) was estimated to be 0.77, 0.86, 0.99, and 0.81 based on analysis of the Dargatz et al. (1997), Hancock et al. (1998b), Smith (1999), and Elder et al. (2000) studies, respectively.

Figure 3-9 shows the estimated distribution for true feedlot prevalence. This distribution suggests that feedlot prevalence is most likely 90%, but it may be as low as 70% or as high as 100%.

These results imply that most, if not all, U.S. feedlots contain one or more *E. coli* O157:H7-infected cattle. Such a result is not surprising given the management—and high turnover rate—of cattle in feedlots. Cattle entering feedlots are typically confined in pens, fed from common feed bunks, and usually shipped to slaughter 3 to 6 months after arrival. Also, feedlot cattle usually originate from multiple locations. Therefore, feedlots hypothetically provide ample opportunity for exposure and transmission of *E. coli* O157:H7 to cattle. The elevated feedlot prevalence estimate from this risk assessment supports such a hypothesis.

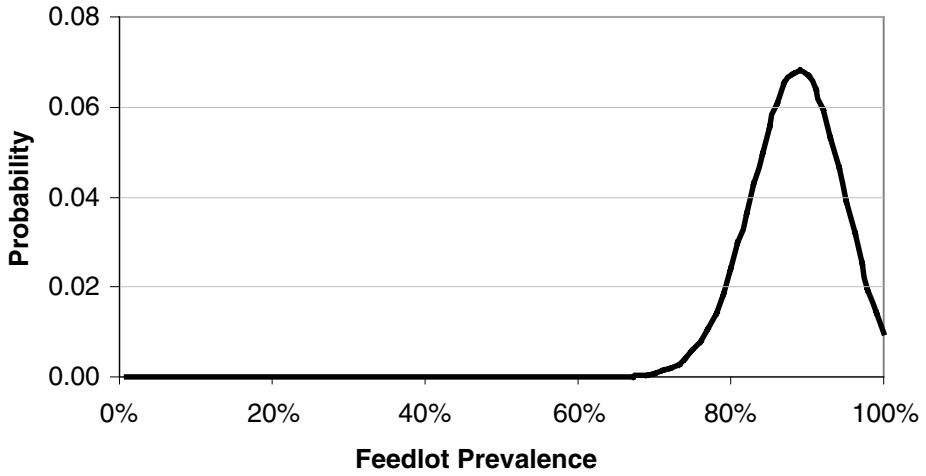

FIGURE 3-9 Resultant uncertainty distribution for true feedlot prevalence after analysis of data in Table 3-6.

Within-Feedlot Prevalence

Within-feedlot prevalence is estimated using the same methods employed for breeding herds.

Apparent Within-Feedlot Prevalence

Population variability. Like within-breeding herd prevalence, within-feedlot prevalence also varies. Figure 3-10 shows the apparent within-feedlot prevalence distribution for 63 infected feedlots (Dargatz et al. 1997). This study included the greatest number of infected feedlots of any published report on U.S. feedlots.

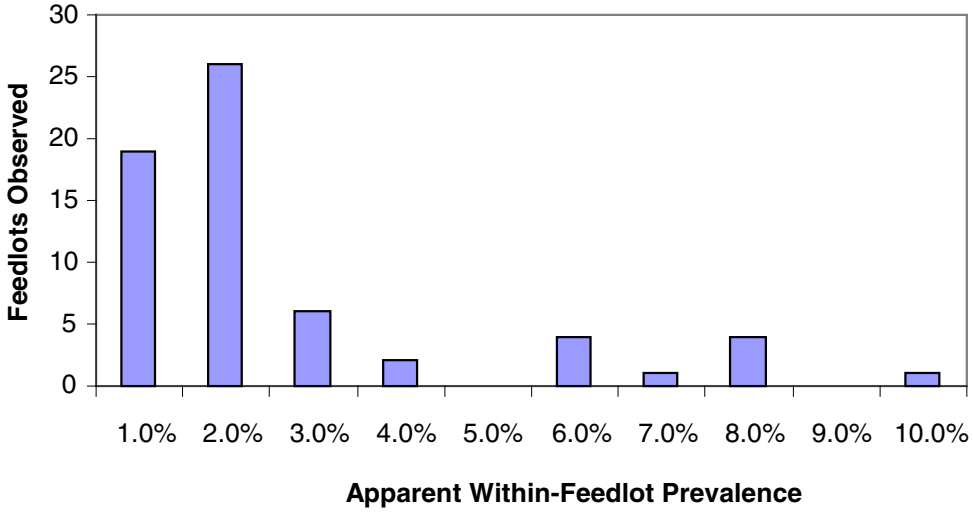

FIGURE 3-10 Evidence on the distribution of within-feedlot prevalence of *E. coli* O157:H7 in infected feedlots (adapted from Dargatz et al. 1997).

As discussed previously, this asymmetric distribution plausibly fits an exponential distribution. The mean and standard deviation of this distribution are 2.7% and 2.2%, respectively. A comparison of this distribution to predictions from an exponential distribution with $\beta = 2.7\%$ also shows some agreement (Figure 3-11). Nevertheless, the hypothesis that the observed and expected results are equivalent is rejected ($\chi^2 = 18.9$, $p<0.05$).

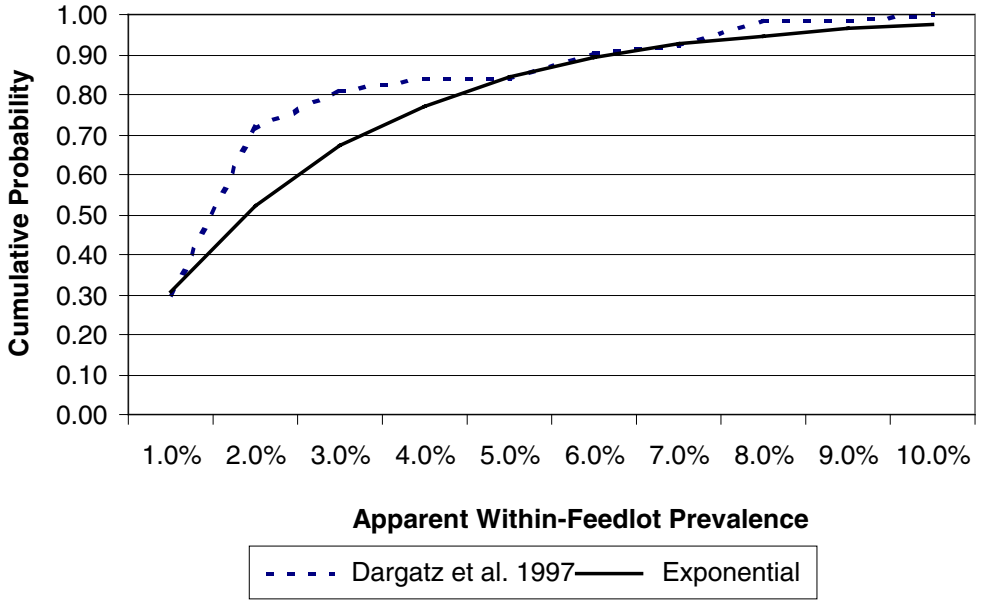

FIGURE 3-11 Comparison of observed and expected cumulative probabilities for within-feedlot prevalence of *E. coli* O157:H7.

Despite the lack of statistical support to conclude that these data fit an exponential distribution, it is assumed that within-feedlot prevalence can be adequately represented with such a distribution. As with breeding cattle studies, most available feedlot data only allow estimation of average within-feedlot prevalence. Therefore, fitting these other data to more complex parametric distributions (e.g., lognormal) is not feasible.

When available data are limited to averages, the principle of Maximum Entropy supports the use of an exponential distribution (Vose 1996). This distribution choice is likely conservative because disagreement between the observed and theoretic distributions tends to occur at lower prevalence levels. Nevertheless, because within-herd prevalence was shown to fit an exponential distribution, such a distribution seems biologically plausible.

Seasonal variability. Most studies of feedlot cattle were completed over limited times of the year. Therefore, evidence of a summer season peak in prevalence is limited for this class of cattle. One Canadian study, which included fed steers and heifers, showed peak prevalence in the summer (Van Donkersgoed et al. 1999). Most U.S. studies completed between June and September report higher *E. coli* O157:H7 prevalence levels than studies completed at other times of the year.

Seasonal variability in within-feedlot prevalence is modeled using the same methods as applied to within-breeding herd prevalence. Although the epidemiology of *E. coli* O157:H7 in cattle is not completely characterized, it seems unlikely that factors (e.g., feed or water

contamination) associated with increased transmission in the warm summer months in breeding cattle are different for feedlot cattle.

Evidence of Apparent Within-Feedlot Prevalence

Five studies provide evidence on apparent within-feedlot (Table 3-7). Dargatz et al. (1997) detected 63 positive feedlots in a national USDA survey. The prevalence of *E. coli* O157:H7-positive cattle in positive feedlots was about 3%. Sampling was conducted between October and December 1994.

TABLE 3-7 Evidence Used to Estimate Within-Feedlot Prevalence

Study	Number Tested in Positive Feedlots	Positive in Positive Feedlots	Apparent Within-Feedlot Prevalence	Lab Methods	Months Sampled
Dargatz et al. 1997	7,560	210	2.8%	0.1 g, SMACct	October–December
Hancock et al. 1998b	1,046	38	3.6%	0.1 g, SMACct	July–November
Hancock et al. 1999	240	14	5.8%	0.1 g, SMACct	November–January, May–June
Smith 1999	3,054	707	23.1%	10 g, IMS	June–September
Elder et al. 2000	254	91	35.8%	10 g, IMS	July–August

Note: g = grams of feces analyzed,
SMACct = sorbitol MacConkey media with cefixime and tellurite, and
IMS = immunomagnetic separation.

Hancock et al. (1998b) found six positive feedlots in three northwestern states. The apparent within-feedlot prevalence was 4%. This study was completed between July and November 1996.

Hancock et al. (1999) studied the prevalence of *E. coli* O157:H7 in the feces of steers and heifers from eight lots at four slaughter plants. When sampling was done just after the cattle were stunned in the slaughter plant, 5.8% of 240 cattle were reported positive. Sampling was conducted in November 1995 to January 1996, and May to June 1996.

Smith (1999) found five positive midwestern feedlots that contained large numbers of positive cattle. The reported apparent within-feedlot prevalence was 23%. The study was conducted from June to September 1999.

Elder et al. (2000) sampled cattle at four midwestern slaughter plants and found 21 positive lots. Within those lots, the prevalence of test-positive cattle was about 36%. This study was conducted in July and August 1999.

Three of these studies used the same sampling and lab methods (Dargatz et al. 1997; Hancock et al. 1998b, 1999). These methods are reportedly 58% sensitive (Sanderson et al. 1995). In contrast, the other studies collected 10-gram samples and used an IMS process followed by microbiologic culture to improve the detection of *E. coli* O157:H7 in fecal samples. As explained previously, this protocol is assumed to be 100% sensitive.

True Within-Feedlot Prevalence

True within-feedlot prevalence data were organized by study months (Table 3-8). No empirical evidence was available between February and April. Therefore, prevalence for these months was calculated using moving averages from the 3 preceding months.

TABLE 3-8 Point Estimates for Monthly True Within-Feedlot Prevalence for Each of Five Studies (Table 3-6). A weighted average for each month was calculated (based on average numbers of samples collected per month per study), and a seasonal average was calculated for the high and low prevalence seasons.

	Weighted Average					
Month	Dargatz et al. 1997	Hancock et al. 1998b	Hancock et al. 1999	Smith 1999	Elder et al. 2000	Average
January			18%			18%
February						10%
March						11%
April						13%
May			4%			4%
June			4%	24%		23%
July		6%		24%	37%	22%
August		6%		24%	37%	22%
September		6%		24%		20%
October	5%	6%				5%
November	5%	6%	18%			5%
December	5%		18%			5%
Weights	2,520	209	48	764	127	
October–May average	(low prevalence season)			9%		
June–September average	(high prevalence season)			22%		
January–December average				13%		

Figure 3-12 illustrates nine random iterations of a Monte Carlo model estimating monthly within-feedlot prevalence. A strong seasonal peak is evident from this graph and is consistent from iteration to iteration.

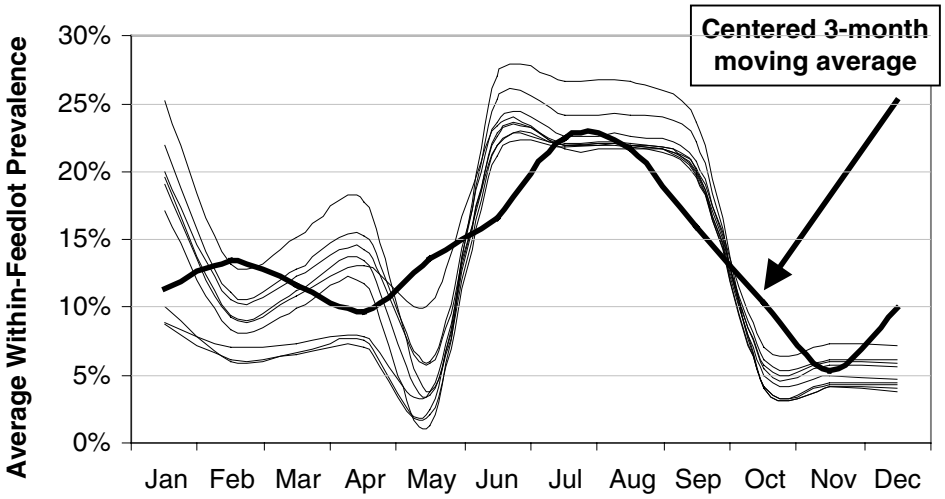

FIGURE 3-12 Estimated average monthly within-feedlot prevalence. This illustrated seasonal trend is based on 1,000 iterations of the model.

Figure 3-13 shows the uncertainty about the low and high prevalence seasonal averages. The mean within-feedlot prevalence is 9% and 22% for the low and high prevalence seasons, respectively. In contrast to the breeding herd analysis (Figure 3-8), the two seasonal distributions are distinctly different, and there is more than a twofold difference between the low and high prevalence seasons for feedlots.

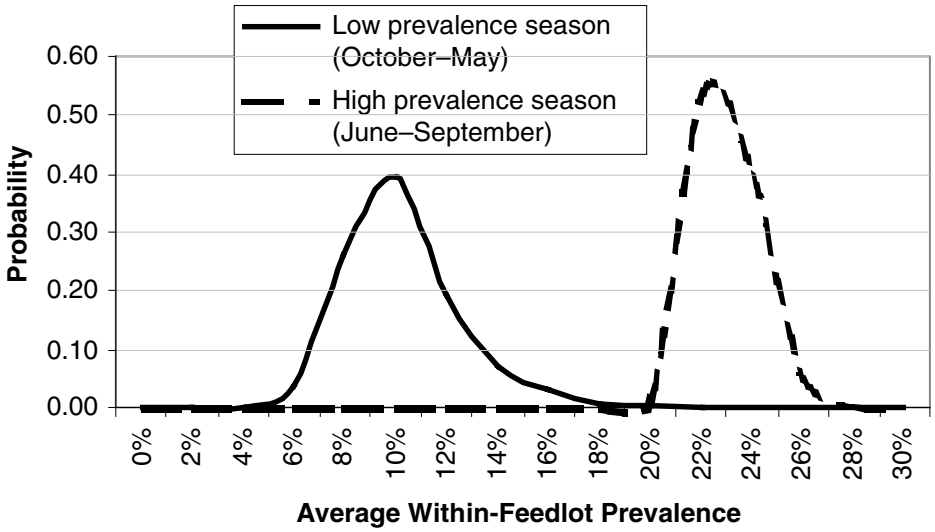

FIGURE 3-13 Uncertainty about low and high prevalence seasons' estimated average within-feedlot prevalence. These distributions were estimated using 1,000 Monte Carlo iterations of the model.

These results imply that within-feedlot prevalence is greater than within-breeding herd prevalence. This difference may be related to cattle age. Feedlot cattle age is typically less than 1 year, while breeding cattle age is over 2 years. A higher prevalence of infection in younger cattle has been previously demonstrated (Hancock et al. 1994; Dargatz et al. 1997; Mechie et al. 1997; Heuvelink et al. 1998; Van Donkersgoed et al. 1999). Acquired or natural immunity may increase with cattle age and result in increased resistance to infection by older cattle. Regardless of cause, the differences in within-feedlot and within-breeding herd prevalence seem consistent with the available evidence.

These results also show that within-feedlot prevalence increases substantially during June to September. At all times of the year, feedlot cattle sent to slaughter are more likely than breeding cattle to be infected. Yet this discrepancy is greatest during the high prevalence season. While there are differences in management between feedlots and breeding herds, the available data do not explain why the seasonal peak is much greater for feedlots than for breeding herds.

Transportation Segment

Transmission of *E. coli* O157:H7 from infected to susceptible cattle may occur when cattle are transported to slaughter. Alternatively, some infected cattle may rid themselves of infection during the period they are being shipped to slaughter. This segment addresses the effect of transportation on prevalence of *E. coli* O157:H7 in feces and hides.

Transportation Effects on Fecal Prevalence

Empirical evidence suggests that there is no dramatic difference in fecal prevalence between the farm and slaughter plant. Rice et al. (1997) collected fecal samples of culled dairy cattle both at the farm and at slaughter. Of 205 samples collected at the farm, 3.4% were *E. coli* O157:H7-positive. Of 103 samples collected at slaughter, 3.9% were *E. coli* O157:H7-positive. Of 89 paired samples (farm and slaughter), 2.2% were positive at both the farm and slaughter, 3.3% were positive at the farm only, and 2.2% were positive at slaughter only.

In a study of New York cull cows (Cornell 1998), 1.3% of 3,323 cull dairy cows were fecal positive for *E. coli* O157:H7 at a slaughter plant. No difference in the average transit time was found between *E. coli* O157:H7-positive cattle and *E. coli* O157:H7-negative cattle (32.6 and 31.7 hours, respectively). Therefore, duration of transportation was not associated with being fecal positive.

In a national study of dairy cattle**,** 2.8% of approximately 600 cows to be culled within the subsequent 7 days were fecal positive for *E. coli* O157:H7 (APHIS-VS-NAHMS 1998). This study also collected fecal samples from over 2,200 dairy cows at livestock markets across the country and found 1.8% of these animals *E. coli* O157:H7-positive.

The data do not suggest that *E. coli* O157:H7 prevalence increases during transport to slaughter. Therefore, no effect from transport is included in the model.

Feedlot cattle are typically shipped directly to slaughter and processed the same day. Therefore, it is reasonable that prevalence is unaffected by transport of this class of cattle. On the other hand, culled breeding cattle are more likely to be shipped to slaughter via livestock markets. This marketing route seemingly increases the elapsed time for shipment. If *E. coli* O157:H7 is transmitted to susceptible cattle during this transport time, the evidence suggests that infected cattle are ridding themselves of infection at a rate equivalent to the transmission rate. In this case, prevalence after shipping remains the same as prevalence before shipping.

Transportation Effects on Hide Contamination

Transit between the farm and slaughter plant may be important in causing changes in hide prevalence. Studies of hide contamination with *Salmonella* suggest an increase in prevalence of hide-contaminated cattle between the farm and slaughter (Puyalto et al. 1997; Cornell 1998).

Data are limited on *E. coli* O157:H7 hide-contaminated cattle. In one study, 1.7% of 240 feedlot cattle at four slaughter plants had hair samples that were *E. coli* O157:H7-positive (Hancock et al. 1999). Paired fecal samples were collected from the animals in this study, and no correspondence between fecal and hide status was found. Elder et al. (2000) collected nonpaired fecal and hide samples from cattle at four slaughter plants. Average fecal prevalence was 28%, yet average hide prevalence was only 11%. Generally, hide-positive lots also contained fecal-positive cattle, but fewer lots were detected from hide sampling. Another study conducted by the American Meat Institute (Bacon et al. 2000) found that 3.6% of 2,245 cattle were hide-positive from samples collected at 12 slaughter plants.

Some researchers hypothesized that the degree of visible soiling of cattle surfaces (e.g., hides, hair) with mud, manure, and/or bedding is correlated with microbial contamination of carcasses (Van Donkersgoed et al. 1997; Jordan 1998). Yet no clear correlation was found. The concentration of *E. coli* Biotype I organisms on carcasses changed very little whether the lot was composed of cattle that had substantial hide soiling or were relatively clean. The implication of this research is that the role of *E. coli* O157:H7 hide contamination in carcass contamination may not be correlated with grossly visible soiling.

Because there are no data on *E. coli* O157:H7 hide-contaminated cattle at the farm and only limited data on hide prevalence at the slaughter plant, the effect of transit time on hide

contamination cannot be examined at this time. The available evidence suggests that fecal prevalence may be a better predictor of carcass contamination than hide prevalence (Elder et al. 2000). If this is the case, then incorporating the effect of hide contamination may be inconsequential. Nevertheless, better hide sampling methods are needed to fully assess the importance of hide prevalence.

Slaughter Plant Intake Segment

Breeding Cattle

Culled dairy and beef cattle arrive at the slaughter plant from their farms of origin after transit on trucks. The majority of these cows and bulls arrive after first being shipped to one or more livestock markets where they are auctioned to the highest bidder and then shipped to slaughter (APHIS:VS:CEAH 1994).

The combined average herd size for dairy and beef herds is approximately 300 cows (NASS 1998). Approximately 25% of cows in dairy herds, and 11% of cows in beef herds, are culled each year (APHIS-VS-NAHMS 1996, 1997). These culling percentages imply that the average herd would ship from 1 to 1.5 cattle per week.

Given the low culling rate per herd, it is reasonable to assume random mixing of breeding cattle at slaughter plants. Such an assumption implies that the prevalence of *E. coli* O157:H7-infected breeding cattle at slaughter is the product of herd prevalence and within-herd prevalence. It also implies that the probability of one cow on the slaughter line being infected is independent of the probability of another cow on the slaughter line being infected. A violation of this assumption would be a group of cows (i.e., 40 cows) from the same farm all sent to slaughter together and then slaughtered one after the other. In this case, the prevalence of infected cows in this group is expected to equal the within-herd prevalence of their herd of origin. Violation of a random mixing assumption is expected to occur rarely.

The number of infected cows and bulls in a group of 40 such animals presented for slaughter was simulated using Monte Carlo techniques. Forty head was a convenient count as it is the capacity of most trucks used to haul cattle to slaughter. Each cow and bull was simulated as an individual. The probability of infection is equal to the product of herd prevalence (H) and average within-herd prevalence (w). The number of infected culled breeding cattle per truckload (B) is simulated as follows:

$$B = \sum_{1}^{40} \text{Binomial}\left[1, H \times \text{Exponential}\left(w\right)\right] \tag{3.6}$$

Within-herd prevalence varies in the population and by season. Average within-herd prevalence (w) is therefore greater for cattle shipped to slaughter during June through September than for cattle shipped during the rest of the year (see Figure 3-8). To model population variability, an exponential distribution—whose only parameter is the mean within-herd prevalence (w)—is used. Monte Carlo simulations then estimate the number of infected cows/bulls in truckloads for the low and high prevalence seasons.

Feedlot Cattle

Steers and heifers arrive at slaughter plants after being transported from their feedlot of origin in a tractor-trailer truck with a capacity of about 40 head. Most steers and heifers (over 90%) are shipped directly from the feedlot to slaughter without going through a livestock market (APHIS:VS:CEAH 1994). Furthermore, these cattle are typically slaughtered together in a group,

although they may be mixed during slaughter with one or more truckloads of cattle from other feedlots.

The manner by which feedlot cattle are marketed does not support the assumption of random mixing used for culled breeding cattle. Instead, feedlot cattle are much more likely to be processed at the slaughter plant in a clustered pattern. Cattle within the same truckload will all have the same probability of infection because they originated from the same pen in a feedlot.

The number of infected feedlot cattle per truckload (F) is simulated as follows:

$$F = \text{Binomial}(1,H) \times \text{Binomial}[40,\text{Exponential}(w)] \quad (3.7)$$

Each truckload is independently determined to be from an infected or noninfected feedlot based on feedlot prevalence (H). If the truck is from an infected feedlot, then the number infected in the truckload is determined based on the appropriate seasonal within-feedlot prevalence (w). Within-feedlot prevalence varies according to the exponential distribution.

Production Module Results

The four critical inputs to the production module are herd prevalence, within-herd prevalence, feedlot prevalence, and within-feedlot prevalence of *E. coli* O157:H7. Herd prevalence is the proportion of all breeding herds that contain one or more infected cattle. Feedlot prevalence is similar, but the reference population is U.S. feedlots. Within-herd (or within-feedlot) prevalence is the proportion of infected cattle within a herd (or feedlot), given that the herd contains one or more infected cattle. Within-herd (or within-feedlot) prevalence is a random variable that modulates by season. Given the available data, these inputs are quantitatively determined.

Analysis of available evidence provides average, 5th, and 95th percentile estimates for these inputs (Table 3-9). Generally, these results demonstrate that *E. coli* O157:H7 prevalence is significantly greater for feedlot cattle than for breeding cattle (e.g., the 95th percentile for herd prevalence is less than the 5th percentile for feedlot prevalence). Similar findings apply to comparisons between within-herd and within-feedlot prevalence, regardless of season.

TABLE 3-9 Statistics for Uncertain Parameters in the Production Module

Model Input	5th Percentile	Mean	95th Percentile
Breeding herd prevalence	55%	63%	72%
Feedlot prevalence	78%	88%	97%
Low prevalence season (October to May)			
Average within-herd prevalence	2%	3%	4%
Average within-feedlot prevalence	6%	9%	14%
High prevalence season (June to September)			
Average within-herd prevalence	3%	4%	5%
Average within-feedlot prevalence	21%	22%	24%

E. coli O157:H7 prevalence was lower for adult cattle than for feedlot cattle in a yearlong Canadian slaughter survey (Van Donkersgoed et al. 1999). In that survey, 2% of breeding cattle and 12% of feedlot cattle were fecal positive.

The model's annual predictions are the same as this Canadian survey. Average breeding cattle prevalence at slaughter is 3% for the June to September period and 2% for the rest of the year (63% × 4% and 63% × 3%, respectively). Feedlot cattle prevalence at slaughter is 19% for the June to September period and 8% for the rest of the year (88% × 22% and 88% × 9%, respectively). Therefore, on an annual basis, the model predicts that 2% of breeding cattle—and 12% of feedlot cattle—are *E. coli* O157:H7-infected just prior to slaughter. Because Van Donkersgoed et al. (1999) used very sensitive test methods, the concordance of this model's results with that survey is especially noteworthy.

The production module simulates cattle entering the slaughter process via truckloads. Therefore, prevalence of infection within truckloads is this model's output and the first input to the slaughter module. The prevalence of infected cattle within truckloads influences the level of *E. coli* O157:H7 contamination that occurs during slaughter. Generally, when the prevalence in a truckload is elevated, contamination during slaughter is also elevated.

For breeding cattle, about 45% of truckloads are predicted to have no infected cattle (i.e., 0% prevalence) during the low prevalence season (Figure 3-14). Because of model input uncertainty, confidence limits for 0% prevalence are between 40% and 52% of truckloads. During the high prevalence season, 35% (±7.5%) of these truckloads are predicted to have no infected cattle. Therefore, truckloads containing infected cattle arrive more frequently at slaughter plants between June and September than at other times of the year.

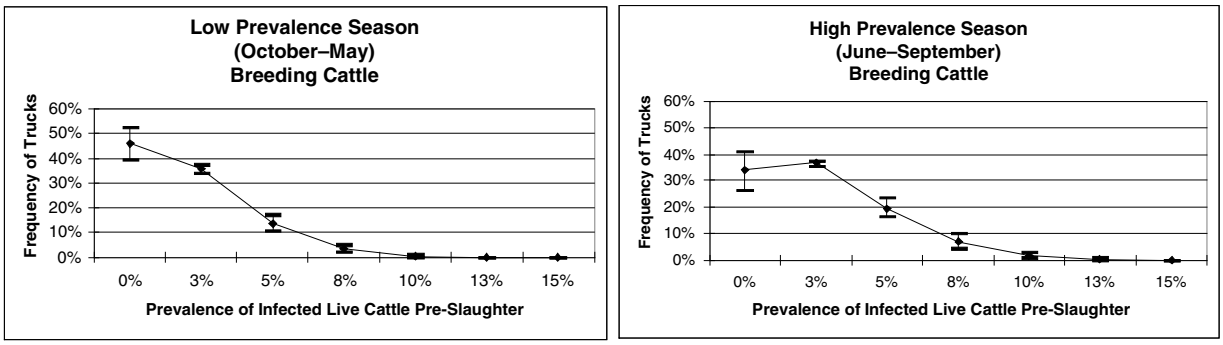

FIGURE 3-14 Comparison of seasonal distributions for prevalence of infected cattle within truckloads of breeding cattle sent to slaughter. Error bars show the 5th and 95th percentiles of uncertainty about frequency of trucks at each prevalence level.

For feedlot cattle, the frequency of truckloads with no infected cattle is about 32% in the low prevalence season and 20% in the high prevalence season (Figure 3-15). Furthermore, there are essentially no trucks with prevalence greater than 30% during the low prevalence season. During the high prevalence season, however, there is a nonnegligible frequency of trucks with greater than 50% prevalence. In fact, there is a 0.1% frequency of trucks with 100% prevalence in the high prevalence season (not shown).

The production model outputs are distributions for cattle prevalence just prior to slaughter. These outputs become the inputs for the slaughter model to follow. The model results predict that feedlot cattle are more likely than breeding cattle to be infected. Furthermore, regardless of cattle type, higher frequencies of infected cattle enter slaughter plants during the June to September period than during the rest of the calendar year. These differences are based on survey data collected in the United States and have been independently verified by data collected in Canada.

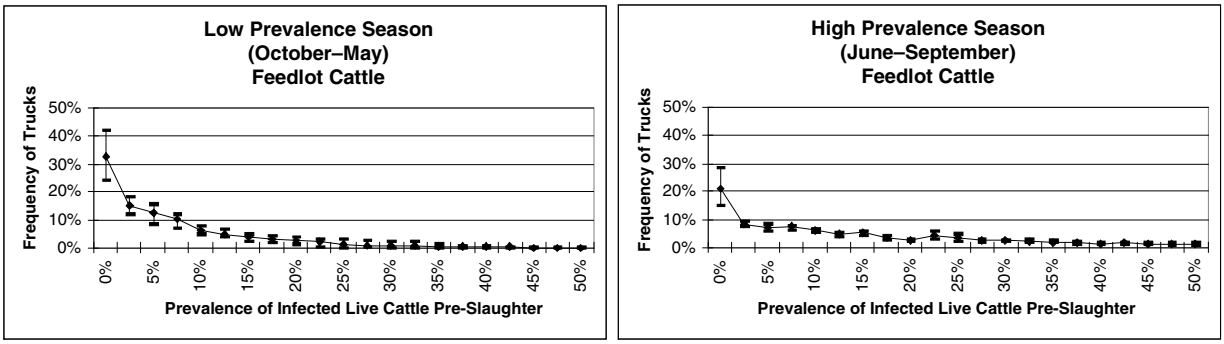

FIGURE 3-15 Comparison of seasonal distributions for prevalence of infected cattle within truckloads of feedlot cattle sent to slaughter. Error bars show the 5th and 95th percentiles of uncertainty about frequency of trucks at each prevalence level.

SLAUGHTER MODULE

The slaughter module estimates the occurrence and extent of *E. coli* O157:H7 contamination as live cattle transition to carcasses, then to meat trim, and finally to aggregates of meat trim in 60-pound trim boxes or 2,000-pound combo bins destined for commercial ground beef production. This module links the production of live cattle to the preparation of ground beef meals by consumers.

Explanation of Scope

Two types of slaughter plants are modeled: those that handle culled breeding cattle and those that handle feedlot cattle. Nevertheless, the same physical plant might slaughter both classes of cattle.

Table 3-10 shows annual slaughter numbers by plant capacity. Forty percent of culled breeding (cow/bull) cattle are slaughtered in large facilities that handle more than 1,000 head per day, while greater than 90% of feedlot (steer/heifer) cattle are slaughtered in such facilities.

TABLE 3-10 Number of Cattle Slaughtered by Type and Plant Capacity, United States, 1997

	Annual Number Slaughtered	
Plant Capacity	Breeding Cattle (Cow/Bull)	Feedlot Cattle (Steer/Heifer)
<1,000 head per day	4.4 million	2.4 million
≥1,000 head per day	3 million	26 million

Source: FSIS 1998.

The model only considers the commercial slaughter and processing of cattle. Although custom slaughter is not explicitly considered in this model, it is assumed to represent a small fraction of ground beef consumed in the United States.

Prevalence distributions of *E. coli* O157:H7 in breeding and feedlot cattle, developed in the production module, serve as inputs to the slaughter module. These distributions provide the number of infected cattle entering a slaughter plant.

Slaughter module outputs are distributions of *E. coli* O157:H7 contamination in combo bins and trim boxes. Breeder and feedlot cattle slaughtering operations are modeled separately, as are

high (June to September) and low (October to May) prevalence seasons. These distributions are inputs to the preparation module, where grinding operations begin the process of converting meat trim in combo bins or boxes into ground beef.

Definition of Key Terms

The following key terms are used throughout this module:

- Carcass refers to an animal that has been killed and had its hide removed.
- Contamination is the presence of *E. coli* O157:H7 on carcass surfaces.
- Trim is a by-product of processing carcasses to create cuts of meat (e.g., steaks, roasts) when the carcasses originate from feedlot cattle. Trim is a primary product that results from deboning carcasses that originate from breeding cattle. Trim consists of both muscle and fat.
- Combo bins are containers that hold 2,000 pounds of meat trim (Gill and Badoni 1997; Biela 1998). The containers are usually cardboard boxes lined with plastic. Many cattle may contribute meat trim to a single combo bin.
- Boxes of meat trim are similar to combo bins but only contain 60 pounds of product.
- Lot is defined as the total number of cattle necessary to fill one combo bin. A single lot may comprise one or more truckloads of cattle.

Slaughter Module Segments

The slaughter module includes seven steps: (1) arrival of live cattle at slaughter plant, (2) dehiding, (3) decontamination following dehiding, (4) evisceration, (5) final washing, (6) chilling, and (7) carcass fabrication (i.e., creation of trim) (Figure 3-16). Although there are other steps that are normally part of the slaughter process (e.g., stunning, carcass splitting), these are not explicitly modeled. Generally, these other steps are incorporated into the seven steps of the model.

Slaughterhouse operating procedures can either facilitate or mitigate the probability of *E. coli* O157:H7 contamination on beef carcasses or trim (Galland 1997). Decontamination steps can significantly reduce the numbers of *E. coli* O157:H7 and other pathogens on the carcass (Bacon et al. 1999). The model assumes that either contamination or decontamination can occur at each step of the process, with the prevalence and extent of contamination increasing if further contamination occurs and decreasing if decontamination occurs. It is possible that a decontamination process is completely effective in eliminating *E. coli* O157:H7 from a carcass, thereby reducing the prevalence of contaminated carcasses. The probability and extent of *E. coli* O157:H7 contamination or decontamination during slaughter are modeled as dependent on status of the incoming animal, type of processing plant, type of equipment and procedures used, efficacy of decontamination procedures, and sanitation processes.

Cattle arrive at slaughter plants (Step 1) via truckloads with variable prevalence of infected cattle. Because slaughter lots may consist of multiple truckloads, each truck's prevalence is estimated in this step, and the total number of infected cattle in the lot is estimated based on the total number of infected cattle on trucks contributing to a combo bin.

Dehiding (Step 2) is the transition from live cattle to carcasses. The process of removing the hide creates the first opportunity for surface contamination of the carcass with *E. coli* O157:H7 and other pathogenic and nonpathogenic microbes. The number of *E. coli* O157:H7 organisms that initially contaminate a carcass depends on the level of infected cattle, the average

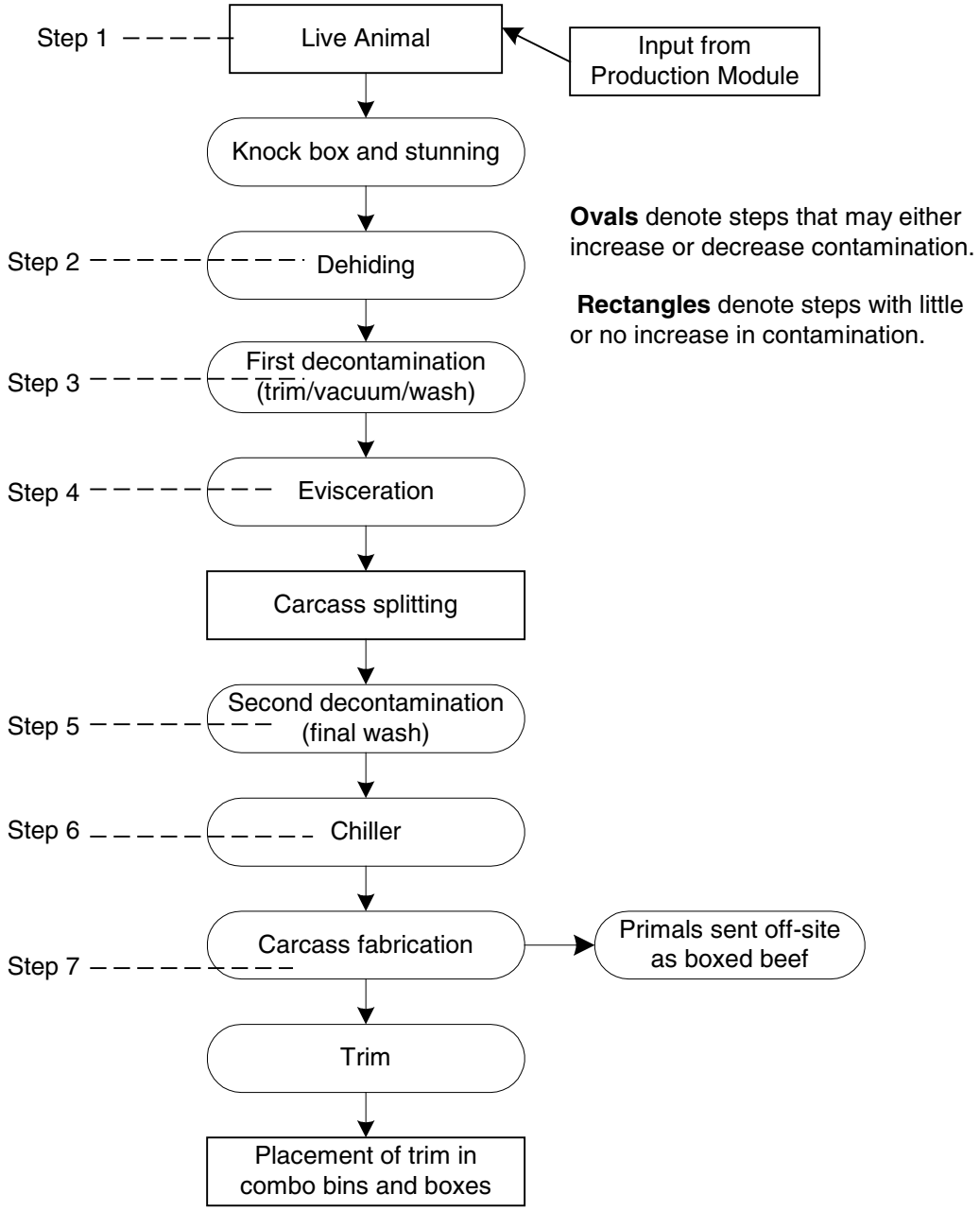

FIGURE 3-16 Steps modeled in the slaughter module.

concentration of *E. coli* O157:H7 per contaminated area, and the total area of a carcass that is contaminated (Galland 1997). Contamination introduced during dehiding can be reduced during decontamination (Step 3). During decontamination, trimming, vacuuming, or washing of the carcass surface can reduce the number of organisms on contaminated carcass surfaces (Prasai et al. 1995).

Evisceration (Step 4) is another opportunity for contamination to be introduced. If any part of the gastrointestinal tract is perforated during the evisceration procedure, *E. coli* O157:H7 contamination of muscle tissue can occur. Carcass splitting and final washing (Step 5) follow evisceration. During final washing, carcasses are washed or steam pasteurized. Washing is the

forceful application of hot or cold water to the surface of the carcass, and pasteurization is the application of steam to the surface of the carcass.

Following final washing, the carcasses move to the chiller (Step 6), where *E. coli* contamination may again increase or decrease. After chilling, the carcasses are fabricated (Step 7). Fabrication involves separating the carcass further into smaller units, trimming these units of excess fat, and—in the case of carcasses from breeding cattle—manually and/or mechanically separating muscle from bone. Feedlot carcasses are typically separated into primal (e.g., quarters) and subprimal units that are used to produce whole-muscle cuts of beef. A by-product of fabricating carcasses from feedlot cattle is meat trim, a product that is mixed and ground to produce ground beef. Because carcasses from breeding cattle produce less valuable whole muscle cuts, greater proportions of these deboned carcasses than carcasses from feedlot cattle contribute to ground beef. The boneless meat trim from one animal is distributed based on fat content into multiple combo bins or boxes, where it is mixed with trim from other cattle. Fabrication can also result in new or additional contamination through cross-contamination of work surfaces.

The following sections describe data and analysis of each slaughter step.

Modeling the Slaughter Process

Arrival of Live Animals (Step 1)

Live cattle are shipped to slaughter via trucks, where they are placed in holding pens prior to entering the knock box. The production module predicts the prevalence of infected cattle per truckload. As mentioned previously, prevalence varies by class of cattle and season. It is assumed that animals arriving at the plant together are processed together.

Number of Trucks Per Lot

The number of trucks that contribute to a slaughter lot depends on the class of cattle, the weight of trim generated per carcass, and the number of combo bins to which carcasses can contribute.

In 1998, 16.2 million steers, 10.6 million heifers, 5.9 million cows, and 0.6 million bulls were commercially slaughtered. Average carcass weights (ACW) for steers, heifers, cows, and bulls were assumed to be 764, 703, 539, and 851 pounds, respectively (NASS 1998). The proportion of carcass weight that amounts to trim (ρ) is 18% for steer/heifer carcasses, 53% for cow carcasses, and 90% for bull carcasses (Duewer 1998; AMIF 1996). These values represent midpoints of uncertainty distributions. Generally, these distributions can range $\pm 20\%$.

Trim from one steer/heifer may go into a variable number of combo bins. The actual number of bins is a function of the number of trim lines operating simultaneously in a particular plant. In steer/heifer slaughter plants, it was assumed that the number of combo bins to which an individual carcass contributes (n) ranged from 2 to 6. In cow/bull plants, this range was 2 to 4. Uncertainty about the most likely number of combo bins per carcass was modeled as a uniform(2,5) and uniform(2,3) for steer/heifer and cow/bull plants, respectively. The ranges and most likely values were modeled using triangular(min, most likely, max) distributions.

The weight of trim a carcass contributes to a single combo bin (ζ) is calculated as follows:

$$\zeta = \frac{\text{ACW} \times \rho}{n} \tag{3.8}$$

The number of carcasses per combo bin equals 2,000 pounds $\div \zeta$. It is assumed that there are 40 cattle per truckload. Therefore, this number of carcasses determines the number of truckloads

of live cattle that contribute to a combo bin (TLD). Consequently, the number of truckloads per lot is as follows:

$$\mathrm{TLD} = \frac{2000}{\zeta \times 40} \tag{3.9}$$

Number of Infected Cattle Per Truck and Lot

Number of infected cattle per truckload originating from breeding herds or feedlots has been previously calculated (Equations 3.6 and 3.7). Trucks in a lot are assumed independent. The total infected cattle in a lot (κ) is the sum of infected cattle from each truck in the lot.

Knock Box and Stunning (Not Modeled)

When it is time for slaughter, the animal is directed out of the holding pen or taken off the truck via a chute to the "knock box," where it is stunned. As the stunned animal falls, it is shackled on one hind leg, raised, and attached by a chain to an overhead rail. A knife is used to slit the throat, and the animal is bled out prior to entering the main floor of the slaughter plant.

Cross-contamination of hides is possible as cattle fall to the floor or come into contact with sides of the chute after previously *E. coli* O157:H7-contaminated cattle have passed through. Additional contamination can occur if cattle emit feces or rumen contents at the knock box (Delazari et al. 1998a, 1998b) or if dirty knives are used (Labadie et al. 1977).

The production module notes the limited data regarding prevalence of *E. coli* O157:H7 on hides and the incomplete analysis of hide sampling method sensitivity. Furthermore, the strongest correlate with carcass contamination is believed to be the fecal status of incoming cattle (Elder et al. 2000). Therefore, the stunning step is not explicitly included in the model. Nevertheless, the contribution of hide contamination to carcass contamination is implicit in the conversion of live cattle prevalence to carcass prevalence within lots.

Dehiding (Step 2)

At this step, cattle enter the main floor of the slaughter plant. Horns and hocks are removed using hydraulic cutters. The udder is removed, the head is skinned, and the hide is cut down the midline, legs, and front shanks.

Contamination Occurrence during Dehiding

The dehiding operation is where a carcass is created. It is at this point that normally sterile muscle and fat tissues on the carcass surface are exposed to microbial contaminants. An individual carcass may be self- or cross-contaminated. If the carcass originates from an animal that is not infected, contamination may occur via aerosol diffusion or contact with contaminated equipment or a contaminated carcass. If the carcass originates from an infected animal, it may be self-contaminated via fecal or hide sources or cross-contaminated by the pathways described for noninfected animals.

The exterior surface of the hide and the environment in the dehiding area are recognized sources of pathogens (Grau 1987). If any cattle are contaminated with *E. coli* O157:H7, cross-contamination can occur via workers' gloves, knives, clothing, or during the changing of the hide-puller from one carcass to the next (Gill 1999). It has been suggested that gross microbial contamination of the carcass is the result of contamination with feces from the hide, hair, hooves and ruptured gut (Siragusa et al. 1998). This contamination can occur as the hide is removed

from the carcass at several steps. For instance, the tail can flip around and create aerosols (Getz 1999) or flip back on the carcass during hide removal. Aerosol contamination can also occur when the hide separates from the carcass (Galland 1997). Hide-removing machinery called up-pullers are possibly more likely to cause aerosol contamination because the hide is being rolled up over the carcass rather than below it.

A transformation ratio (TR) relates the frequency of contaminated carcasses to the frequency of infected cattle in a lot. To estimate the fraction of carcasses contaminated during dehiding, evidence from a study in four slaughter plants is used (Elder et al. 2000). In this study, cattle fecal prevalence and carcass prevalence were measured during July and August 1999. In lots showing evidence of *E. coli* O157:H7 in cattle or on carcasses, 91 of 307 cattle (30%) and 148 of 312 carcasses at dehiding (47%) were *E. coli* O157:H7-positive. Therefore, a higher frequency of contaminated carcasses than infected cattle was detected in this study. Very sensitive testing methods were used in this study, and the results are assumed indicative of the relationship between live cattle and carcass prevalence. However, this study was completed during the summer months, and inferences drawn from it are most applicable to the high prevalence season (June to September).

It is possible that proportionally fewer carcasses are contaminated during the low prevalence season (October to May). Incoming prevalence of infected cattle is generally lower in this season. Consequently, the probability of a carcass becoming contaminated may be reduced because less contamination enters the slaughter plant environment. In a study of 12 slaughter plants conducted in September and October 1999, the prevalence of *E. coli* O157:H7 hide-contaminated cattle was 3.56%, while the *E. coli* O157:H7 prevalence of contaminated carcasses was 0.44% (Bacon et al. 2000). These results suggest a lower frequency of contaminated carcasses than contaminated cattle entering slaughter plants.

During the high prevalence season, TR is estimated from the Elder et al. (2000) data. Uncertainty about TR is modeled by incorporating these data into beta distributions (i.e., TR = $\frac{\text{beta}\,(148 + 1{,}312 - 148 + 1)}{\text{beta}\,(91 + 1{,}307 - 91 + 1)}$). Using the average TR for the high prevalence season, the frequency of contaminated carcasses is estimated to be 160% of the prevalence of incoming infected cattle.

During the low prevalence season, TR is modeled as a mixture of the beta distributions based on the Elder et al. (2000) data and a uniform distribution with a minimum approaching 0 and a maximum of the summer TR. Therefore, more uncertainty is modeled about TR during this season. Using the average TR for the low prevalence season, the frequency of contaminated carcasses is estimated to be 120% of the prevalence of incoming infected cattle.

The number of contaminated carcasses per lot (C_d) depends on the number of infected cattle per lot (κ) and TR:

$$C_d = \kappa \times \text{TR} \tag{3.10}$$

It is assumed that C_d is a random Poisson variable (i.e., $C_d \sim \text{Poisson}\,[\kappa \times T]$).

Level of Contamination Per Carcass

The number of *E. coli* O157:H7 organisms on a contaminated carcass at dehiding is calculated from the estimated density per cm^2 and the total contaminated surface area.

No studies have reported the density of *E. coli* O157:H7 contamination at the dehiding step. Bell (1997) measured densities of generic *E. coli* on carcasses and found 2 logs CFU/cm^2 contamination if the carcass came into contact with feces or a contaminated hide, and 1 log

CFU/cm^2 contamination due to cross-contamination (e.g., aerosols, hands, equipment, or contact with a contaminated carcass).

More relevant data regarding *E. coli* O157:H7 density on carcasses are available from the FSIS (1994) national baseline survey of slaughter plants. In this survey, a 60-cm^2 surface area was sampled from each of 2,081 chilled carcasses originating from feedlots. Four (0.2%) carcasses were *E. coli* O157:H7-positive, and enumerated densities were reported (Table 3-11).

TABLE 3-11 Enumeration of *E. coli* O157:H7 Densities on Positive Carcasses Detected by FSIS USDA (1994)

CFU/cm^2	Number of Samples	Percent of Total
<0.030	2	50
0.030 to 0.300	0	0
0.301 to 3.000	2	50
Total	4	100

Elder et al. (2000) found 6 (2%) of 330 chilled carcasses positive for *E. coli* O157:H7 using very sensitive test methods. This prevalence is substantially greater than that found in the FSIS survey (0.2%) and suggests that some contaminated carcasses were not detected in the latter survey. A ratio of these results (i.e., 0.2% ÷ 2%) suggests that about 10% of contaminated carcasses were detected in the FSIS survey and that about 90% of contaminated carcasses were below the limit of detection for that survey. This ratio (*S*) is modeled as follows:

$$S = \frac{\text{beta}(4+1, 2081-4+1)}{\text{beta}(6+1, 330-6+1)} \tag{3.11}$$

Additionally, of the four carcasses reported *E. coli* O157:H7-positive in the FSIS (1994) survey, two (50%) of the positive samples had densities below the measurable limit of 0.03 CFU/cm^2. Consequently, an average of about 5% of all contaminated carcasses would be expected to have values above 0.03 CFU/cm^2.

The proportion of carcasses contaminated below the measurable limit (*L*) is modeled as $L = S + (1 - S) \times [2 \div (4 + 1)]$. In other words, the proportion of carcasses below the measurable limit includes those carcasses not detected and those detected carcasses with unmeasurable densities. A value of one is added to the total enumerated carcasses to adjust for bias (Vose 1996).

The initial number of *E. coli* O157:H7 organisms on contaminated carcasses introduced during dehiding (*I*) is modeled as a cumulative frequency distribution (Table 3-12). The minimum number of *E. coli* O157:H7 organisms predicted from this distribution is 1 organism on the total contaminated surface area. The maximum number of *E. coli* O157:H7 organisms is assumed to be 3 *E. coli* O157:H7 per cm^2. Although the amount of contamination is variable, there is also uncertainty about *S* and the number of *E. coli* O157:H7 organisms observed in the FSIS survey (1994).

TABLE 3-12 Inputs Used to Model the Number of *E. coli* O157:H7 Organisms per Contaminated Carcass

O157 Organisms per cm^2	Cumulative Frequency (%)
0.03	$L = S + (1 - S) \times [2 \div (4 + 1)]$
Uniform(0.3,3.0)	$L + (1 - S) \times [2 \div (4 + 1)]$

There is no evidence regarding the total contaminated surface area (A) on carcasses. The total outside surface area (TSA) of steer/heifer, cow, and bull carcasses is about 32,000, 23,000, and 37,000 cm^2, respectively (McAloon 1999). Arbitrarily, the minimum area that contamination might be spread across is assumed to be 30 cm^2 (based on the measurable detection threshold). Hypothetically, the maximum area that contamination might be spread across for each carcass type is the total outside surface area. Nevertheless, initial model runs showed that contaminated surface areas greater than 3,000 cm^2 produced results that were infeasible in comparison with FSIS ground beef sampling data (see Appendix A). Therefore, uncertainty about the total contaminated surface area is modeled as $A = 10^{\text{triangular}[\log10(30),\log(300),\log(3000)]}$.

The total number of organisms on a contaminated carcass at dehiding (OCC_d) is calculated as follows:

$$OCC_d = I \times A \tag{3.12}$$

Therefore, the maximum number of organisms on a contaminated carcass predicted by this model is 3 organisms/cm^2 × 3,000 cm^2, or 9,000 *E. coli* O157:H7 organisms, and the minimum is 1 *E. coli* O157:H7 organism per contaminated carcass.

First Decontamination (Step 3)

Following removal of the hide, one or more decontamination steps may be applied depending on the amount of visible foreign matter on the carcass. Knife trimming is used to remove visible spots of fecal contamination greater than 1 inch in diameter. Spot steam vacuuming is used to remove visible spots of fecal contamination that are less than 1 inch in diameter (FSIS 1996). Increasingly, plants are rinsing carcasses with hot water and a variety of organic acids prior to evisceration.

Any one of the three decontamination steps can reduce existing *E. coli* O157:H7 on the carcass (Bacon et al. 1999; Galland 1997). The effectiveness of knife trimming is highly variable (Prasai et al. 1995), and cross-contamination through the knife cuts can occur if inadequate knife sterilization methods are used. Sheridan et al. (1992) and Smeltzer et al. (1998) have identified equipment such as knives, gloves, and aprons as reservoirs of bacteria in the slaughterhouse.

Two experimental studies have measured the reduction of *E. coli* on inoculated beef resulting from rinsing ingesta and manure from the carcass. Gill (1999) reported that carcass rinses reduced generic *E. coli* counts by 0.32 log CFU/cm^2. Dorsa et al. (1997) reported a 0.7 log CFU/cm^2 reduction with a water rinse.

For decontamination to be effective, the procedure needs to be applied to the affected area. While visible signs of foreign matter can be readily identified and removed, bacterial colonies themselves are not directly observable. Thus, there is variability associated with the decontamination step actually encountering bacterial colonies as well as variability in any reductions in contamination. To capture this variation, the reduction from decontamination (D1) was modeled using a triangular distribution with a minimum value of 0 logs, an uncertain most

likely value ranging from 0.3 to 0.7 logs, and an uncertain maximum value ranging from 0.8 logs to 1.2 logs.

Evisceration (Step 4)

During evisceration, the ventral midline of the carcass is split and the gastrointestinal tract is removed. The remaining organs (bladder, lungs, heart, etc.) are also removed from the carcass in this stage.

Studies indicate that evisceration is usually carried out with minimal contamination (Bell 1997; Gill et al. 1996a; Gill et al. 1996b). Nevertheless, it was assumed that *E. coli* O157:H7 contamination of muscle tissue could occur if any part of the gastrointestinal tract was perforated during the sawing of the brisket (i.e., chest) and other procedures. In addition, the gastrointestinal tract of some animals may be weaker and easily tear during evisceration (Galland 1997).

Brewer (1999) suggests that perforation along the gastrointestinal tract potentially occurs in 1 out of every 100 carcasses. The probability of this event (ε) is independent of the *E. coli* O157:H7 status of the animal from which the carcass originates. Uncertainty about this probability uniformly ranges from 0% to 2%.

If the intestine of a non-*E. coli* O157:H7-infected animal ruptures during evisceration, then self-contamination of that carcass is assumed not to occur. The number of carcasses that are contaminated at evisceration (C_e) is calculated as follows:

$$C_e = \kappa \times \varepsilon \tag{3.13}$$

It is assumed that C_e is a binomial distribution (i.e., C_e ~ binomial(κ, ε). If a rupture occurs in a carcass from an infected animal, then the number of *E. coli* O157:H7 that contaminate this carcass is predicted as described for dehiding ($OCC_e = I \times A$).

Carcass Splitting (Not Modeled)

At this step, the carcass is sawed in half, the tail is removed, and excess fat is trimmed away from each side. Hypothetically, the carcass might become contaminated with *E. coli* O157:H7 if a clean carcass comes into contact with contaminated machinery, hands, or other contaminated carcasses during splitting. No data are available on this type of contamination.

Second Decontamination (Step 5)

The second decontamination step occurs after carcass splitting. Different procedures for this decontamination step are used depending on the size of the plant.

Knife trimming of visibly contaminated meat occurs in both large and small plants after the carcass is split. Spot steam vacuuming may also be used in some plants. Many plants have implemented at least two decontamination interventions, such as steam pasteurization and carcass rinses, that are effective in reducing pathogens on carcasses (*Federal Register* 1998). Decontamination of carcasses can occur as visible fecal or ingesta spots are removed from the carcass via knife and/or steam vacuuming. During the carcass rinse step, *E. coli* O157:H7 can be reduced or redistributed over the entire carcass (Bell 1997). Steam pasteurization of carcasses can significantly reduce contamination, if properly done (Gill 1998).

It was assumed that large plants typically use a steam pasteurization process with four steps: (1) four sides of beef are enclosed in a stainless steel pressure chamber, (2) vertical blowers remove excess surface water, (3) steam is applied for 5 to 15 seconds, and (4) a cold water rinse

is applied. The effectiveness of this equipment depends on the temperature of the steam and the duration it is applied.

It was assumed that small plants typically use a hot water rinse, sometimes supplemented with organic acids. The effectiveness of hot water rinsing is assumed equivalent to that described for decontamination Step 1 (D1).

Efficacy of steam pasteurization has been assessed. Phebus et al. (1997) found a 3.53 ± 0.49 log CFU/cm^2 reduction in *E. coli* O157:H7 on inoculated carcasses. Gill (1998) reported up to a 2 log CFU/cm^2 reduction for generic *E. coli* from pasteurizing at 105.0°C (221.0°F) for 6.5 seconds. Nevertheless, if the carcass was not clean and dry before steam pasteurization, there was little effect from steam pasteurization. Other studies have shown reductions in prevalence of *E. coli* O157:H7-contaminated carcasses from steam pasteurization (Nutsch et al. 1997, 1998).

Kastner (1998) reported that steam pasteurization was effective in reducing *E. coli* O157:H7 only if the temperature was 93.3°C (200.0ºF) for 6 seconds or more. Phebus (personal communication 1999) suggested that the standard industry practice uses 87.8ºC (190.0ºF) steam for 6 to 8 seconds.

Given standard industry behavior and available evidence, variability in steam pasteurization efficacy (i.e., D2 for large plants) was modeled using a triangular distribution with a minimum value of 0 logs, an uncertain most likely value of 0.5 to 1.5 logs, and an uncertain maximum value of 1.51 to 2.5 logs.

Chiller (Step 6)

After the sides of beef are decontaminated for the second time, they go into a blast air chiller for 24 to 48 hours. FSIS regulations require chilling deep muscle (6 inches) to 10.0°C (50.0ºF) within 24 hours and to 7.2°C (45.0ºF) within 36 hours (NACMCF 1993). Sides of beef are automatically or manually spaced on overhead rails within the chiller and are periodically sprayed with water. Occasionally distilled water, chlorine, or a lactic acid solution may also be used. After chilling, the sides are unloaded, graded, and sorted.

Growth or decline of *E. coli* O157:H7 on the surface of carcasses is largely a function of time and temperature. Fluctuations in chiller temperature, or the outright failure to adequately chill carcasses, may enable growth. Gill and Bryant (1997) reported that generic *E. coli* counts increased by 0.25 logs in one slaughterhouse and decreased by 1.34 log CFU/cm^2 in another slaughterhouse. Dorsa (1997) found a 1.2 log CFU/cm^2 increase in *E. coli* O157:H7 on carcasses stored for 2 days in the chiller at 5.0°C (41.0ºF). Although deep tissue mass cools slowly, it is generally sterile and thus not necessarily a problem (Bailey and Cox 1976; Gill 1979).

Growth or decline is assumed only to occur on carcasses where *E. coli* O157:H7 is already present before entering the chiller. Changes to *E. coli* O157:H7 populations on carcasses during chilling (CH) are modeled using a normal distribution with an uncertain mean ranging from –0.5 to 0.5 logs and a standard deviation of 1 log. Therefore, the most likely effect from chilling is that there is no change in the *E. coli* O157:H7 count on carcasses, yet substantial changes can occur with nonnegligible frequency (e.g., 2 or more logs of growth can occur in 2.5% of lots).

Carcass Fabrication (Step 7)

Carcasses move from the chiller to the fabrication floor, which is usually maintained at 10ºC (50ºF). The fabrication step is complicated and typically involves many plant personnel operating on different lines to process different parts of the carcass.

In feedlot cattle plants, sides of beef enter the fabrication step on overhead rails where they are cut into primals (major cuts of beef) and subprimals (minor cuts of beef). Most primal cuts

are taken from the rail using a hook and knife. Leftover trim moves on conveyers to either the combo bins or to a vacuum packaging area. The trim is either put into combo bins to which dry ice is added prior to shipment, or it is vacuum packaged and put into boxes maintained at a temperature between 0°C and 2ºC (32.0°F to 35.6ºF).

The fabrication area in slaughter plants is cleaned at the end of each day with a hot water power washer that may contain sanitizers. Larger pieces of meat and trim are periodically picked off equipment and carted away. Knives, chain-mail aprons, and gloves are washed with hot water.

During the cutting and deboning operations, contamination is possible from environmental sources and contaminated sides of beef. The major source of contamination is likely to be the surface of incoming carcasses. Freshly cut surfaces of meat may be further contaminated when in contact with processing surfaces, equipment, conveyer belts, cutting surfaces, knives, gloves, and aprons during slaughter (Charlebois et al. 1991). Gill et al. (1999) found that despite a stringent sanitation regimen, and inspection by the national regulatory authority and internal plant quality assurance staff, *E. coli* O157:H7 persisted and proliferated on conveyer equipment in obscure areas that continued to contaminate the meat-contacting surface.[1]

Cross-contamination can occur via workers' hands and the commingling of trim (Newton et al. 1978). Fabrication rooms are typically kept at 10ºC (50ºF), but lapses may occur and the higher temperatures that result enable microbial growth. Gill (1996) has demonstrated that the practice of cooling meat trim with dry ice in combo bins is generally effective in preventing *E. coli* O157:H7 growth. Scanga et al. (2000) found no difference in the concentration of *E. coli* O157:H7 across fat content. Prasai et al. (1995) found no difference in concentrations of *E. coli* O157:H7 between hot deboning and cold deboning.

Minimal data are available on frequency and amounts of *E. coli* O157:H7 contamination during the fabrication process. Three studies report increases in general bacterial growth during this process. Hardin et al. (1995) report increased bacterial contamination on beef surfaces during the trimming process even with the use of sterile utensils under experimental conditions. Charlebois et al. (1991) sampled four locations within fabrication and concluded that the deboning operations resulted in the highest final count of fecal coliforms on boneless beef. Specifically, it was found that of 378 samples, the percent of samples that had more than 500 fecal coliform/cm^2 increased from 0.8% to 6.6%. A study in four plants found increases in generic *E. coli* contamination during fabrication ranging from 0 to 2 logs CFU/cm^2 (Gill 1999).

The data suggest that the fabrication step might result in increased *E. coli* O157:H7 populations on meat trim. The quantitative evidence is limited. Therefore, the fabrication effect is indirectly estimated.

This indirect estimate results from the output of the grinder segment in the preparation module. FSIS ground beef sampling data for 2000 were used to set upper and lower limits for ground beef contamination (see Appendix A). Simulations of the slaughter segment that resulted in expected contamination greater than the upper limits were discarded as implausible. Simulations of the slaughter segment that resulted in expected contamination below the lower limits had additional contamination added. This additional contamination represents the effect of fabrication (F).

During the low prevalence season, the model estimates the average effect from fabrication to be 0.33 logs. This effect can range from 0 logs to 1.5 logs because of uncertainty in the model

[1]The cleaning regimen involved "the cleaning of the carcass breaking equipment, the removal of gross detritus by brushing and sweeping, washing with high pressure sprays of cold water, coating with a foaming detergent, washing with high pressure sprays of hot water, and treatment of the cleaned equipment with a chlorine sanitizer" (Gill et al. 1999).

inputs and methods. A 0.33 log increase implies that contamination levels entering combo bins are more than doubled (i.e., $10^{0.33} = 2.1$) as a result of fabrication. During the high prevalence season, the increase from fabrication is 0.22 logs, with a similar range of uncertainty. Therefore, the average effect of fabrication is estimated to be substantial from this model. This conclusion supports the suggestion of some researchers that fabrication is a critical step for *E. coli* O157:H7 transfer and amplification within the slaughter process.

Contamination in Combo Bins and Boxes

Contamination from a Single Carcass

For each carcass contaminated during the dehiding step (but not during evisceration), the number of *E. coli* O157:H7 organisms (E_d) after fabrication is calculated as follows:

$$E_d = (\mathrm{OCC_d} \times 10^{-DC1} \times 10^{-DC2} \times 10^{CH} \times 10^{F}) \quad (3.14)$$

In other words, the number of organisms initially on the carcass ($\mathrm{OCC_d}$) is proportionally reduced by the log reductions predicted by *DC*1 and *DC*2, proportionally increased or decreased during the chilling step (*CH*), and proportionally increased during fabrication (*F*) (Table 3-13).

TABLE 3-13 Illustrative Example for Calculating the Number of Organisms Remaining on a Single Carcass Following Fabrication. In this scenario, contamination only occurs at dehiding. Therefore, the evisceration step is omitted in this example.

Steps	Symbol	Example Value	Comments
Dehiding (2)	$\mathrm{OCC_d}$	100 organisms	
First decontamination (3)	DC1	0.5 logs	$10^{-0.5} = 0.32$, Step 3 results in a 68% reduction in organisms.
Second decontamination (5)	DC2	1 log	$10^{-1} = 0.1$, Step 5 results in a 90% reduction in organisms.
Chilling (6)	CH	0 logs	$10^{0} = 1$, Step 6 results in no change in organisms.
Fabrication (7)	F	1 logs	$10^{1} = 10$, Step 7 results in a tenfold increase in organisms.
Organisms remaining (Equation 3.4 through 3.13)	E	32 organisms	

For a carcass contaminated only at evisceration, the number of *E. coli* O157:H7 organisms (E_e) remaining after fabrication is calculated similarly:

$$E_e = (\mathrm{OCC_e} \times 10^{-DC2} \times 10^{CH} \times 10^{F}) \quad (3.15)$$

Because evisceration contamination occurs after the first decontamination step in the process, the first decontamination step does not influence the final number of organisms remaining on this carcass.

For a carcass contaminated at both the dehiding and evisceration steps, the number of *E. coli* O157:H7 organisms (E_f) remaining on the carcass after fabrication is calculated as follows:

$$E_f = \left[(\mathrm{OCC_d} \times 10^{-DC1}) + \mathrm{OCC_e}\right] \times 10^{-DC2} \times 10^{CH} \times 10^{F} \quad (3.16)$$

Given any E and the surface area contaminated (A), the density of *E. coli* O157:H7 contamination (η) on a carcass is calculated as follows:

$$\eta = \frac{E}{A} \tag{3.17}$$

The amount of *E. coli* O157:H7 is signified as η_d, η_e, η_b to indicate the type of carcass contamination.

All of the *E. coli* O157:H7 contamination is assumed to be on the surface of the carcass. Seventy-five percent of a steer/heifer carcass surface area is estimated to end up in ground beef (McAloon 1999). For cow/bull carcasses, approximately 90% of the surface area goes into trim. The number of cm^2 per pound of trim (φ) depends on the total surface area, the percent of surface area that becomes trim, and the total weight of trim. It is calculated as

$$\varphi = \frac{\text{TSA}_\text{a}}{\text{ACW} \times \rho}, \tag{3.18}$$

where TSA_a is the total surface area adjusted for the percent trim.

For each carcass, the pounds of trim a single carcass contributes to a combo bin are previously calculated as ζ (Equation 3.8). Therefore, the total cm^2 placed into a combo bin per carcass is the product of ζ and φ.

Some fraction of the total surface area placed into a combo bin from a carcass is contaminated with *E. coli* O157:H7. This fraction depends on the total cm^2 placed in the combo bin ($\zeta \times \varphi$) and the probability of a contaminated cm^2 ($\text{A} \div \text{TSA}$). The number of contaminated cm^2 a carcass contributes to a combo bin (CC) is distributed as follows:

$$\text{CC} \sim \text{Binomial}(\zeta \times \varphi, \frac{\text{A}}{\text{TSA}}) \tag{3.19}$$

The total number of *E. coli* O157:H7 organisms a carcass contributes to a combo bin (CBO) depends on the number of *E. coli* O157:H7 organisms on the carcass per contaminated cm^2 and the total contaminated cm^2 entering the combo bin. It is distributed as follows:

$$\text{CBO} \sim \text{Poisson}(\eta \times \text{CC}) \tag{3.20}$$

Contamination from Entire Lot

A combo bin consists of the contributions from many carcasses. *E. coli* O157:H7 contamination contributed to a combo bin can come from cattle that are contaminated at dehiding, or evisceration, or at both steps. The probability that a carcass is contaminated at evisceration depends on it being from an infected animal. In contrast, carcasses contaminated at dehiding may either originate from infected or noninfected cattle. The probability that a dehiding-contaminated carcass is also from an infected animal is unknown but is assumed uniform(0,1). If a lot consists of carcasses that are contaminated at dehiding and at evisceration (as predicted by C_d and C_e), then the number of carcasses contaminated at both sites (C_b) is predicted as binomial [minimum(C_d,C_e),uniform(0,1)]. Therefore, the number of carcasses contaminated only at dehiding is $C_d - C_b$, and the number of carcasses contaminated only at evisceration is $C_e - C_b$.

The amount of *E. coli* O157:H7 contamination in a combo bin depends on the number of contaminated carcasses and the amount of contamination each carcass contributes. The total

amount of *E. coli* O157:H7 contributed by dehiding-contaminated carcasses ($TCBO_d$) is calculated as follows:

$$TCBO_d = \sum_{0}^{C_d - C_b} \text{Poisson}(\eta_{d_i} \times CC_i) \quad (3.21)$$

Similarly, the total amount of *E. coli* O157:H7 contributed by eviscerator-contaminated carcasses ($TCBO_e$) and those carcasses contaminated at both steps ($TCBO_b$) are calculated as follows:

$$TCBO_e = \sum_{0}^{C_e - C_b} \text{Poisson}(\eta_{e_i} \times CC_i) \text{ and} \quad (3.22)$$

$$TCBO_b = \sum_{0}^{C_b} \text{Poisson}(\eta_{b_i} \times CC_i) \quad (3.23)$$

The total *E. coli* O157:H7 in a combo bin (TCBO), therefore, is calculated as follows:

$$TCBO = TCBO_d + TCBO_e + TCBO_b \quad (3.24)$$

Boxes

Boxes are 60-pound versions of combo bins. Therefore, the number of *E. coli* O157:H7 in a box (TBXO) is calculated as follows:

$$TBXO = \text{Poisson}\left(TCBO \times \frac{60 \text{ pounds}}{2000 \text{ pounds}}\right) \quad (3.25)$$

Slaughter Module Results

Outputs from the slaughter module are distributions describing the frequency of *E. coli* O157:H7 in combo bins (and boxes) generated during high and low prevalence seasons for cow/bull and steer/heifer slaughter plants. These outputs become inputs to the preparation module, in which the contents of combo bins (i.e., trim) are processed to produce ground beef.

Combo Bins

Figure 3-17 shows distributions of *E. coli* O157:H7 contamination in combo bins generated from the slaughter of cows and bulls. These results were estimated from 100 simulations of the model. During the low prevalence season, the mean frequency of combo bins containing no *E. coli* O157:H7 is 94%, but this frequency might range between 88% and 97% because of uncertainty in model inputs. During the high prevalence season, an average of 92% (ranging from 85% to 97%) of combo bins contain no *E. coli* O157:H7. Therefore, an average of 6% and 8% of combo bins generated from breeding cattle are contaminated with 1 or more *E. coli* O157:H7 organisms during the low and high prevalence seasons, respectively. Furthermore, the average combo bin contains 2 and 3 *E. coli* O157:H7 organisms in the low (October to May) and high (June to September) prevalence seasons, respectively.

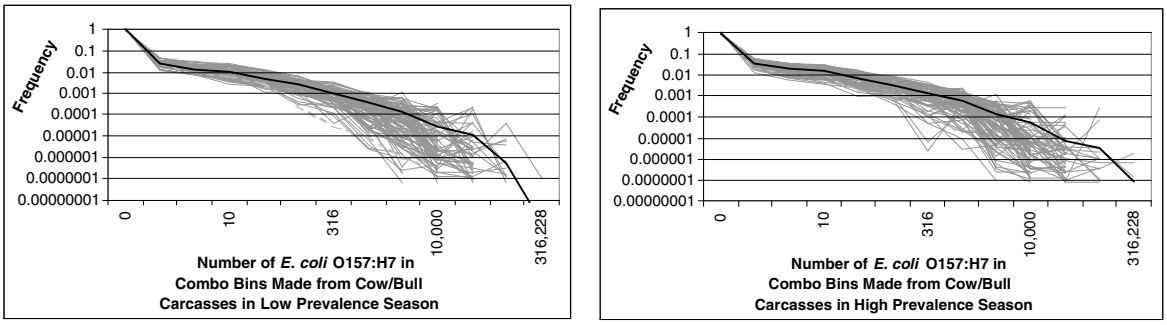

FIGURE 3-17 Comparison of seasonal distributions for number of *E. coli* O157:H7 in combo bins constructed from the slaughter of breeding (cow/bull) cattle. Dark lines are the mean distributions for each season.

Figure 3-18 shows distributions of *E. coli* O157:H7 contamination in combo bins generated from the slaughter of steers and heifers. These results were also estimated from 100 simulations of the model. During the low prevalence season, an average of 77% (ranging from 55% to 97%) of combo bins generated from steer/heifer carcasses contained no *E. coli* O157:H7. During the high prevalence season, 57% (ranging from 42% to 83%) of these combo bins contained no *E. coli* O157:H7. Therefore, an average of 23% and 43% of combo bins contain 1 or more *E. coli* O157:H7 organisms during the low and high prevalence seasons, respectively. Furthermore, the average combo bin contains 13 and 41 *E. coli* O157:H7 organisms in the low and high prevalence seasons, respectively.

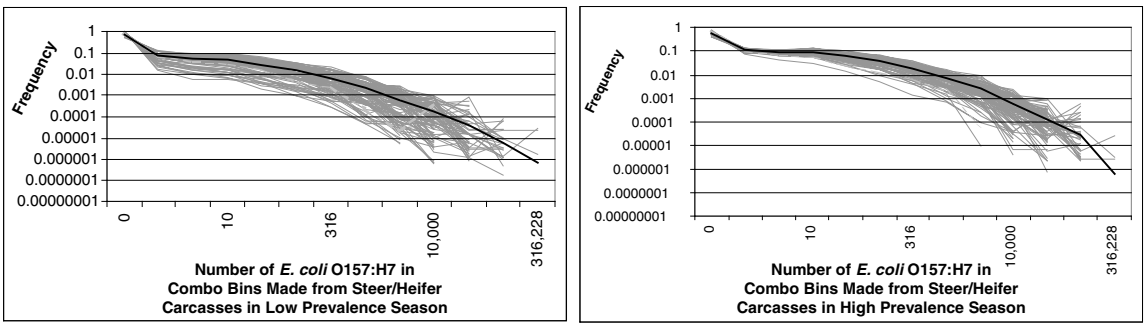

FIGURE 3-18 Comparison of seasonal distributions for number of *E. coli* O157:H7 in combo bins constructed from the slaughter of feedlot (steer/heifer) cattle. Dark lines are the mean distributions for each season.

These results show that prevalence and contamination levels in combo bins increase during the high prevalence season. These seasonal differences in combo bin contamination reflect the trends in prevalence of infected cattle entering slaughter. As noted previously, the influence of season is much greater for feedlot cattle than for breeding cattle. For combo bins generated from feedlot cattle, prevalence of contaminated combo bins increases nearly twofold, and average contamination levels increase over threefold, during the high prevalence season. Therefore, ground beef generated from steer/heifer combo bins is likely to be substantially more contaminated during the June to September period than ground beef produced during the other months of the year.

These results also show that combo bins generated from steer/heifer carcasses are more likely to be contaminated than those generated from cow/bull carcasses. On average, there is about a

fourfold greater prevalence of contaminated combo bins generated from steer/heifer carcasses compared with those generated from cow/bull carcasses during the low prevalence season. This difference is over fivefold during the high prevalence season. These differences reflect the differences noted for incoming live cattle prevalence between these two classes of cattle.

Boxes

Figure 3-19 shows distributions of *E. coli* O157:H7 contamination in meat trim boxes generated from the slaughter of cows and bulls. These results were estimated from 100 simulations of the model. During the low prevalence season, an average of 99% of boxes (ranging from 97% to 100%) contain no *E. coli* O157:H7. During the high prevalence season, an average of 98% of boxes contain no *E. coli* O157:H7. Therefore, about 1% and 2% of boxes generated from breeding cattle are contaminated with 1 or more *E. coli* O157:H7 organisms during the low and high prevalence seasons, respectively. Regardless of season, the average box concentration is much less than 1 *E. coli* O157:H7 organism in the low and high prevalence seasons.

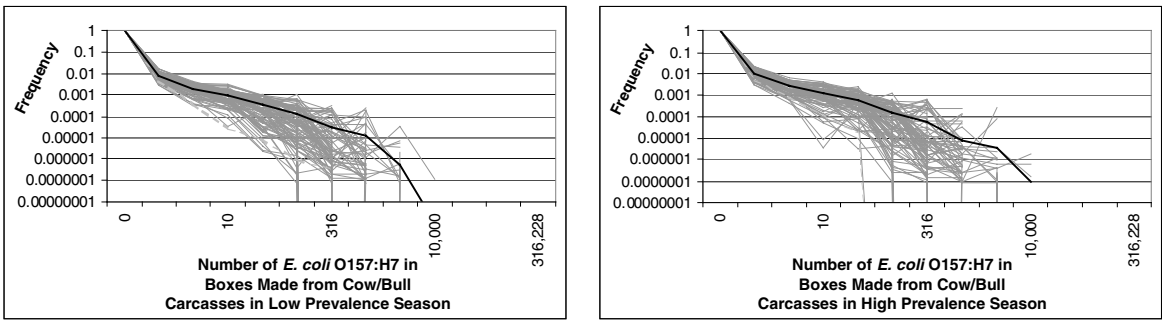

FIGURE 3-19 Comparison of seasonal distributions for number of *E. coli* O157:H7 in 60-pound boxes constructed from the slaughter of breeding (cow/bull) cattle. Dark lines are the mean distributions for each season.

Figure 3-20 shows distributions of *E. coli* O157:H7 contamination in meat trim boxes generated from the slaughter of steers and heifers. These results were also estimated from 100 simulations of the model. During the low prevalence season, an average of 94% (ranging from 87% to 99%) of boxes generated from steer/heifer carcasses contained no *E. coli* O157:H7. During the high prevalence season, 87% (ranging from 79% to 97%) of these boxes contained no *E. coli* O157:H7. Therefore, about 6% and 13% of boxes contain 1 or more *E. coli* O157:H7 during the low and high prevalence seasons, respectively. The average box contains almost 0.5 and 1 *E. coli* O157:H7 organisms in the low and high prevalence seasons, respectively.

By definition, boxes consist of less meat trim than combo bins. Consequently, prevalence and levels of *E. coli* O157:H7 in these aggregates of meat trim are less than observed for combo bins. However, the number of ground beef servings generated from boxes is correspondingly reduced. Therefore, the risk to consumers from ground beef generated from boxes is not likely to be much different from the risk from ground beef generated from combo bins. Seasonal trends and cattle class differences noted for combo bins are also noted for boxes.

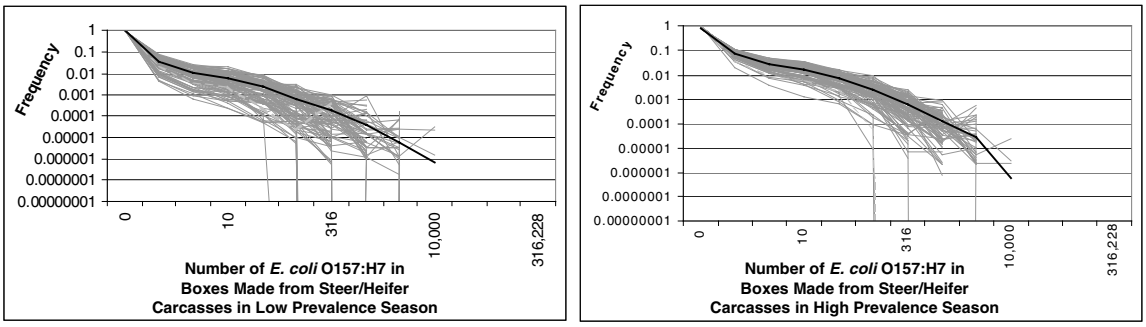

FIGURE 3-20 Comparison of seasonal distributions for number of *E. coli* O157:H7 in 60-pound boxes constructed from the slaughter of feedlot (steer/heifer) cattle. Dark lines are the mean distributions for each season.

PREPARATION MODULE

The preparation module estimates the occurrence and extent of *E. coli* O157:H7 contamination in consumed ground beef servings. This module also characterizes the consumption of ground beef servings by age of consumer and location of meal.

Explanation of Scope

The preparation module simulates the annual consumption of approximately 18 billion ground beef servings. It considers the effects of handling and cooking on the amount of *E. coli* O157:H7 in contaminated servings. Ground beef is consumed in many forms. Typical forms are hamburger patties, ground beef as a formed major ingredient (e.g., meatballs and meat loaf), and ground beef as a granulated ingredient (e.g., ground beef in spaghetti sauce). The model focuses on the first two forms. Because granulated ground beef has a relatively large surface area compared with volume, the effect of cooking on this product is considered to be similar to intact beef products. Intact beef products are considered to be safe after cooking (NACMCF 1997). Furthermore, products incorporating granulated ground beef are often subjected to further cooking. Consequently, these types of products are assumed to have no viable *E. coli* O157:H7 organisms and are not modeled.

Although cross-contamination could be a potential contributor for contamination of ground beef product, cross-contamination of ground beef products is not modeled. An analysis of potential pathways in which ground beef could be contaminated by food service workers or other foods—or alternatively, pathways in which ground beef could contaminate other products—is beyond the scope of this risk assessment. Currently, quantitative modeling of cross-contamination in foods is hampered by a dearth of evidence. Furthermore, cross-contamination pathways are potentially complex, and each pathway may require as much data regarding growth dynamics and cooking effect as the primary product of interest. The model, however, can serve as a starting point for analyzing the effects of cross-contamination on human exposure to *E. coli* O157:H7.

Definition of Key Terms

The following key terms are used throughout this module:

- Servings are defined as an "eating occasion" within the 1994–1996, 1998 Continuing Food Survey of Individual Intakes (CSFII) database. The amount of ground beef consumed per eating occasion varies by age of the consumer and location where the meal was consumed (i.e., at home versus away from home).
- Exposure refers to the amount of contamination that is consumed in a serving.
- Home is used when servings are prepared and served in a home environment.
- Away from home is used when servings are prepared and served in an institutional environment. This is often referred to as "HRI" (hotels, restaurants, and institutions).
- Transportation refers to nonrefrigerated transport of product from a retail or wholesale establishment to its place of preparation and consumption.
- Retail refers to establishments, such as grocery stores or butcher shops, that sell ground beef for home consumption.
- Wholesale refers to establishments that serve as distributors for HRI for away from home consumption.
- High prevalence season refers to June through September.
- Low prevalence season refers to October through May.

Preparation Module Segments

The preparation module consists of six primary steps (Figure 3-21). Five of these steps explicitly model growth, decline, or dispersion of *E. coli* O157:H7 contamination: (1) grinding, (2) storage during processing by the retailer or distributor, (3) transportation home or to HRI, (4) storage at home and "away from home" (i.e., HRI), and (5) cooking. Step 6 models the amount of ground beef consumed, which varies depending on the age of the consumer and the location where the meal was consumed.

Inputs to this module consist of the frequency and extent of *E. coli* O157:H7 contamination in combo bins and boxes estimated in the slaughter module. The preparation module output consists of a single exposure distribution depicting the frequency and extent of *E. coli* O157:H7 contamination consumed in a year.

Grinding (Step 1) transforms combo bins and boxes into ground beef. Combo bins are processed in large commercial facilities, and boxes are typically processed in smaller settings such as grocery stores.

In Step 1, multiple combo bins or boxes are combined, mixed, and extruded to produce finished ground beef with a specific fat content. For example, a combo bin consisting of 90% lean trim can be mixed with another combo bin of 50% lean trim to make a grinder load of 70% lean ground beef. Although the extent of *E. coli* O157:H7 contamination does not increase during the grinding process because of temperature controls, contamination from a single combo bin or box can be dispersed during grinding to contaminate many individual ground beef servings. Consequently, assuming a constant frequency of contaminated combo bins, the number of combo bins that contribute to a grinder load determines whether the grinder load is contaminated. Once ground beef is produced, it can be shaped into patties or packaged in bulk containers and shipped for eventual consumption. Some beef is also ground at retail or institutional sites. This beef consists of 60-pound boxes, in addition to trim generated in the facility and beef that has already been ground at a grinding facility.

Storage conditions at retail or wholesale (Step 2) provide an opportunity for *E. coli* O157:H7 levels to (a) increase as a result of time and temperature abuse or (b) decrease as a result of the effects of freezing ground beef (Ansay et al. 1999; Sage and Ingham 1998). Ground beef is

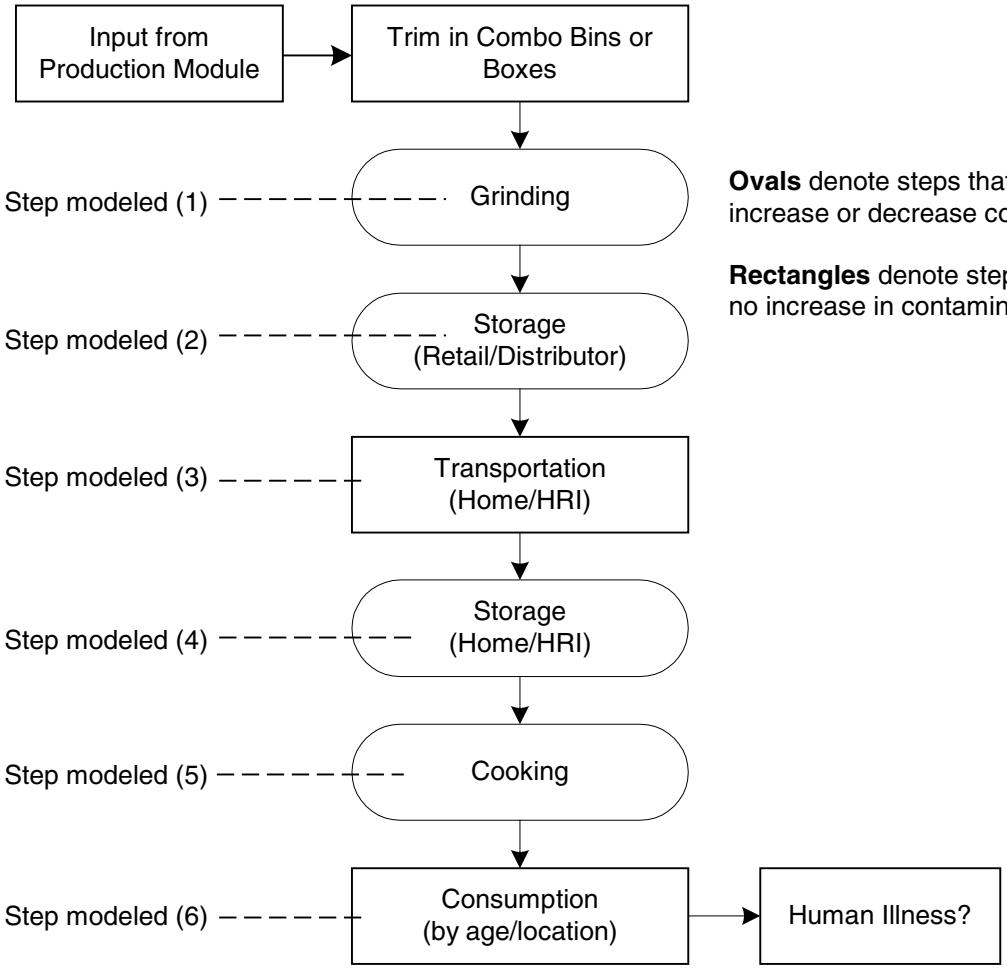

FIGURE 3-21 Steps modeled in the preparation module.

subject to a variety of temperatures during storage and handling conditions, depending on its site of production and ultimate use. These conditions at home and in HRI can significantly increase the numbers of *E. coli* O157:H7 in ground beef (Marks et al. 1998; Buchanan and Bagi 1994; Walls and Scott 1996).

Step 3 models the effects of time and temperature during transportation on the level of *E. coli* O157:H7 after the ground beef is purchased.

Step 4 models the storage of ground beef in the freezer or refrigerator prior to its preparation and consumption and provides another opportunity for increases or decreases in *E. coli* O157:H7 contamination in ground beef servings.

Ground beef is usually cooked prior to consumption (Step 5). Cooking can significantly reduce *E. coli* O157:H7 in ground beef servings (D'Sa et al. 2000; Juneja et al. 1997; Jackson et al. 1996). The model uses final internal product temperature data from a commercial food temperature database (Audits International 1999) to determine the level of reduction in *E. coli* O157:H7 contamination in ground beef servings.

Step 6 models consumption of *E. coli* O157:H7-contaminated ground beef servings, taking into consideration the age group of the consumer (i.e., 0 to 5, 6 to 24, 25 to 65, and 65+ years of age) and where the meals were consumed (i.e., at home or away from home).

The following sections describe data and analysis for each preparation step.

Modeling the Preparation Process

Input from the Slaughter Module

The slaughter module provides distributions for *E. coli* O157:H7 contamination in combo bins and boxes by season for breeding (cow/bull) and feedlot (steer/heifer) cattle slaughter plants. Combo bins can be mixed across slaughter plant type (i.e., combo bins originating from cow/bull plants can be mixed with combo bins originating from steer/heifer plants). Combo bins are characterized by the "leanness" of the ground beef. Requirements for specific fat content in ground beef dictate which combo bins are mixed.

Grinding Beef Trim (Step 1)

Ground beef produced in the United States is sold to the general public through retail establishments (41%) or to HRI through wholesale distributors (59%) (APHIS:VS:CEAH 1994). Retail establishments may use coarse ground beef and mix it with trimmings produced in-house. They may also buy "case ready chubs" (plastic tubes filled with 5 to 10 pounds of ground beef). About 22% of retail ground beef contains at least some retail trimmings (APHIS:VS:CEAH 1994). Of the ground beef used in HRI, 98% percent comes directly from grinder establishments.

E. coli O157:H7 contamination in beef trim generated in the slaughter module is used as an input to the grinding step in the preparation module. As noted in the slaughter module, beef trim is generated either from cows and bulls or from steers and heifers. Although individual cows and bulls generate more trim than individual steers and heifers, the slaughtering of greater numbers of steers and heifers results in about 60% of domestic beef trim coming from this source (Table 3-14). As noted previously, about 15% of beef is imported and either used by itself or mixed with domestic product. It is assumed that this product is similar to domestically produced product. Figure 3-22 depicts the three types of beef used to make ground beef (i.e., beef trim from cows/bulls or steers/heifers, imported beef trim, or ground beef).

TABLE 3-14 Percent of Meat Trim by Types of Cattle (Cows, Bulls, Steers, and Heifers)

Carcass Type	Average Carcass Weight (lbs.)	Percent Trim	Annual Slaughter (Million Head)	Total Meat Trim (Million lbs.)	Percent of Trim by Class
Cow	539	53%	6.9	1,970	40% Cow/Bull
Bull	851	90%	0.7	540	
Steer	764	18%	17.4	2,390	60% Steer/Heifer
Heifer	703	18%	11.2	1,420	

The model combines combo bins of three types of beef trim (Figure 3-22) to simulate a grinder load of beef. It includes beef that may be blended with other ground beef after initial grinding. For example, beef from two separate grinder loads of 10,000 pounds representing five combo bins each could be further mixed together to create a grinder load of 20,000 pounds. The number of combo bins (NCB) that are mixed together to create a grinder load ranges uniformly from 2 to 15 (Smith 1998, personal communication).

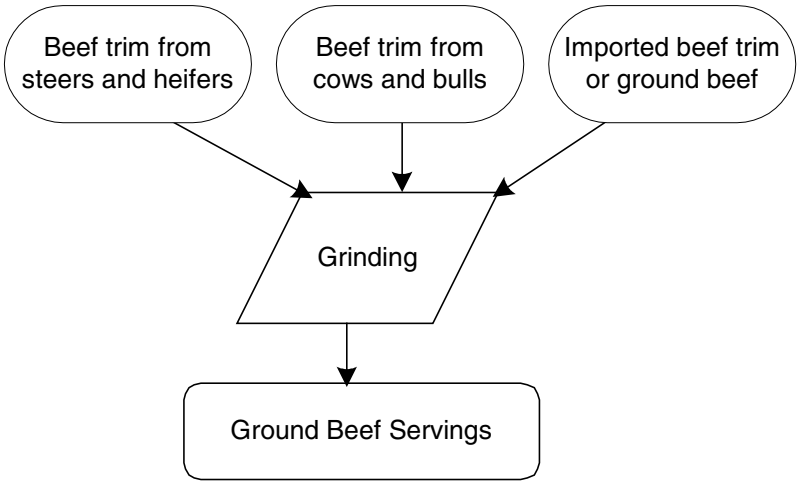

FIGURE 3-22 Inputs to grinding (Step 1).

Retail ground beef is modeled as coming from one to seven 60-pound boxes of beef trim. Equation 3.26 represents the process as modeled for combo bins:

$$\underset{\text{in grinder load}}{E.coli\ \text{O157:H7}} = \sum_{2}^{NCB} \text{Discrete}\left[\left(\underset{\text{in combo bin}_i}{E.coli\ \text{O157:H7}}\right),(P_i)\right]_{\infty}^{\infty} \qquad (3.26)$$

The discrete distribution in Equation 3.26 consists of two arrays. The first array represents various contamination levels that may occur in a combo bin, and the second array represents the corresponding probability of the occurrence of each *E. coli* O157:H7 contamination level.

After *E. coli* O157:H7-contaminated beef trimmings have been ground, the next load may be contaminated unless the grinder has been thoroughly cleaned and sanitized. Farrell et al. (1998) reported that ground beef inoculated with 6 logs of *E. coli* O157:H7 per gram resulted in contamination of a grinder with approximately 3 logs per cm^2. Washing the grinder lowered the contamination to about 1 log per cm^2. Sanitizing the grinder further lowered the contamination to less than 1 log per cm^2. Initial contamination of ground beef in a grinder with 2 logs of *E. coli* O157:H7 per gram followed by cleaning and sanitizing with chlorine resulted in no detection of *E. coli* O157:H7 organisms. This "carryover" *E. coli* O157:H7 contamination between grinder loads was not modeled because (1) Farrell et al. (1998) show that even without cleaning and sanitizing between grinder loads, there was still a 3 log reduction in the number of *E. coli* O157:H7 organisms; (2) the number of *E. coli* O157:H7 organisms present in grinder loads is very low and is therefore assumed not to contribute significantly to contamination of the next grinder load; and (3) such carryover contamination could potentially increase the number of contaminated grinder loads but would result in a corresponding decrease in the number of *E. coli* O157:H7 in the previous grinder load.

Storage and Transportation (Steps 2 through 4)

From the time that ground beef is produced until it is prepared and consumed, it is stored under varying conditions. Ground beef product may be produced at the slaughter establishment, shipped immediately to retail, purchased shortly thereafter, and prepared. Product could also be produced from beef trim that was sent to a grinding establishment where it was held before it was shipped to a wholesaler and stored for additional time. It could then be purchased in bulk by an HRI establishment and stored in a freezer before refrigeration, thawing, and final preparation.

In addition to variations in storage time, variations in fat content of the ground beef, strain of *E. coli* O157:H7, and packaging can also contribute to growth or decline in the number of *E. coli* O157:H7 organisms in ground beef.

Modeling Growth

Increase or decrease in the number of *E. coli* O157:H7 organisms in ground beef is based on the time in which ground beef is stored at certain temperatures. This risk assessment models growth of *E. coli* O157:H7 in ground beef based on three assumptions:

1. All areas of a product are at the same temperature. In reality, the outside of the product would reach a stable temperature first, with the inside of the product reaching a stable temperature last. To construct corresponding cooling curves, however, would require data and assumptions about the frequency of product thickness, its correlation to storage temperature, and the corresponding times of storage. The result would be a much more complicated model that would not be more useful because the underlying assumptions would not be well supported.
2. All *E. coli* O157:H7 strains exhibit the same growth characteristics in any ground beef product. This risk assessment model further assumes that temperature during storage and handling is the only significant variable to predict growth. Although factors other than temperature are known to influence the growth of *E. coli* O157:H7 (Buchanan and Bagi 1994), this simplifying assumption is necessary to permit modeling. Nevertheless, variability in growth is modeled based on the available evidence.
3. Lag period duration in any one stage is affected by temperatures in previous stages. The lag period (the time prior to cell division) duration is modeled as a cumulative percentage that begins at 100% and decreases as product is subjected to varying temperatures at different stages along the farm-to-table continuum. Although it is reasonable to assume that *E. coli* O157:H7 organisms exposed to significantly different storage conditions would need additional time to adjust to those conditions before entering into a rapid growth phase, this assumption avoids the complications of making additional assumptions about when to restart calculations for lag period duration. As a result, this assumption in the risk assessment may result in an overestimation of the increase in the number of *E. coli* O157:H7 organisms in ground beef during storage and handling.

Estimation of the effect of storage temperatures on the growth and decline of *E. coli* O157:H7 is based on two types of data:

- consumer and retail time and temperature data from commercial food temperature databases (Audits International 1999), and
- predictive microbial growth data for *E. coli* O157:H7 in ground beef from published scientific literature (Ansay et al. 1999; Marks et al. 1998; Sage and Ingham 1998; Jackson et al. 1996; Walls and Scott 1996; Buchanan and Bagi 1994).

Several studies show the effect of temperature on *E. coli* O157:H7 levels in ground beef. Palumbo (1997) reported that *E. coli* O157:H7 grows at 8°C (46.4°F) but not at 5°C (41°F). Gill and Bryant (1997) reported a decline of generic *E. coli* as a result of freezing. Ansay et al. (1999) tested the effects of refrigeration over time. In this study, storage of ground beef patties at 2°C for 4 weeks resulted in a 1.9 log reduction of *E. coli* O157:H7, and storage at –2°C for 4 weeks resulted in a 1.5 log reduction. Freezing (–20°C) for 1 year resulted in a 1 to 2 log reduction while tempering (at 15°C for 4 hours) increased the log reduction brought about by storage at –2°C. Sage and Ingham (1998) tested the effects of freezing (–20°C, 24 hours) and thawing on *E. coli* O157:H7 in ground beef and found a wide range in freeze-thaw sensitivity, with a decrease in *E. coli* O157:H7 levels from 0.62 to 2.52 logs per gram.

Predictive microbiological models have been developed for *E. coli* O157:H7 in ground beef under various storage conditions. These microbiological models predict the growth and decline of *E. coli* O157:H7 given environmental parameters including time, temperature, pH, and salinity. One set of equations was developed by Buchanan and Bagi (1994) and was later incorporated into the Pathogen Modeling Program (PMP) available from the Agricultural Research Service. Another set of equations has been developed by Marks et al. (1998).

Walls and Scott (1996) compared predictions from the PMP with observations of *E. coli* O157:H7 growth in ground beef and concluded that the PMP "offers reasonably good predictions of growth in raw ground beef" (p. 1,335). Table 3-15 compares the predictions from the Marks et al. (1998) equations with the predictions from the PMP (Buchanan and Bagi 1994) and the Walls and Scott (1996) observations. Both sets of predictions gave similar results, although the Marks et al. (1998) equations gave closer predictions to lag time and generation time.

TABLE 3-15 Comparison of Pathogen Modeling Program (PMP) with Marks et al. (1998) Equations Using Walls and Scott (1996) Observations

Growth Conditions		Generation Time (hours)			Lag Period Duration (hours)			T1000 (hours)—Time for 3 Log Increase in *E. coli* O157:H7 Organisms		
Temp	pH	Walls and Scott (1996)	PMP	Marks et al. (1998)	Walls and Scott (1996)	PMP	Marks et al. (1998)	Walls and Scott (1996)	PMP	Marks et al. (1998)
12°C	5.7	6.00	3.80	3.62	16.20	30.50	26.99	76.70	68.50	63.19
12°C	6.3	3.90	3.20	3.62	2.78	27.20	26.99	38.60	59.50	63.19
20°C	5.7	1.50	1.00	1.11	2.08	8.34	6.83	17.60	18.30	17.96
20°C	6.3	1.30	1.00	1.11	1.25	7.54	6.83	14.40	17.30	17.96
35°C	5.7	0.40	0.30	0.38	1.23	1.53	1.52	5.00	4.80	5.29
35°C	6.3	0.40	0.30	0.38	1.05	1.40	1.52	5.10	4.60	5.29

The Marks et al. (1998) equations used temperature as the only parameter. Since a single parameter model requires less information, and since these equations also included adjustments for the variability inherent in the system, these are the ones used in the model. Given a temperature (τ) in °C, the following sets of equations are used to predict growth of *E. coli* O157:H7 in ground beef:

Lag period duration (LPD) is calculated as follows:

$$\ln(\text{LPD}) = 9.98 + [-2.69 \times \ln(\tau)] \tag{3.27}$$

ln(LPD) has a standard deviation of 0.27. Consequently, the distribution is modeled as $\ln(\text{LPD}) \sim \text{normal}\{9.98 + [-2.69 \times \ln(\tau), 0.27]\}$.

Generation time (GT) is calculated as follows:

$$\ln(\text{GT}) = 7.03 + \{-6.31 \times \ln[\ln(\tau)]\} \tag{3.28}$$

ln(GT) has a standard deviation of 0.16. Consequently, the distribution is modeled as $\ln(\text{GT}) \sim \text{normal}\left(7.03 + \{-6.31 \times \ln[\ln(\tau)]\}, 0.16\right)$.

The maximum population density (MPD) (e.g., the maximum number of *E. coli* O157:H7 organisms) is calculated as follows:

$$MPD = TMD + (-0.014 \times \tau) \quad (3.29)$$

The theoretic maximum density (TMD) at refrigeration temperatures was estimated by Marks et al. (1998) to be about 10 logs. Walls and Scott (1996) also demonstrated growth in ground beef up to 10 logs. However, the maximum growth of *E. coli* O157:H7 achievable in ground beef is also thought to be a function of the total microbial population density in the food. Such a phenomenon has been demonstrated for *Salmonella* where the suppression of growth of all microorganisms in the food occurred when the total microbial population achieved the MPD characteristic of the food (Jameson 1962). This effect has also been reported for *S. aureus*, *L. monocytogenes*, and *Carnobacterium* spp. (Buchanan and Bagi 1997; Duffes et al. 1999; Nilsson et al. 1999; Ross and McMeekin 1991; Grau and Vanderlinde 1992).

Because maximum growth of *E. coli* O157:H7 possible in a food depends on the population of all microbes, and the population of other microbes in ground beef varies, it is assumed that the TMD varies. A triangular distribution is used to model this variability, where the minimum TMD is assumed to be 5 logs, the maximum TMD is assumed to be 10 logs, and the most likely TMD is uncertain but can range uniformly from 5 to 10 logs.

From Marks et al. (1998), the MPD has a standard deviation of 0.15 and is thus modeled as follows:

$$MPD = \text{normal}\{\text{triangular}[5, \text{uniform}(5,10), 10] + (-0.014 \times \tau), 0.15\} \quad (3.30)$$

Output from a Monte Carlo simulation of these equations overlaps most of the observations from Walls and Scott (1996) with three exceptions: the prediction of the lag period duration (1) for a temperature of 12°C (54°F) at a pH of 6.3, (2) for a temperature of 20°C (68°F) with a pH of 5.7, and (3) for a temperature of 20°C (68°F) with a pH of 6.3. In each case, the equations overestimate the lag period duration. Nevertheless, the T1000 times, which incorporate both the LPD and GT, overlap the Walls and Scott (1996) observations for all conditions.

Continued research of the effect of various storage condition combinations (e.g., pH, moisture, packaging, freezing, refrigeration, thawing) on *E. coli* O157:H7 levels in ground beef products would allow construction of better predictive microbial models. Incorporation of such models into risk assessment is further dependent on studies to develop frequency distributions for various storage conditions.

Modeling Storage Temperature

As noted in Figure 3-22, this model includes the effects of storage temperature on the increase or decrease of *E. coli* O157:H7 in ground beef at three steps: (1) retail or wholesale storage, (2) transportation to the location of preparation (i.e., home or HRI), and (3) storage before cooking. Temperatures for all three steps are based on internal product temperatures of ground beef taken on nearly 1,000 samples (Audits International 1999). Table 3-16 shows numbers of occurrences of storage temperatures above 45°F.

The model assumes that *E. coli* O157:H7 levels do not increase at refrigeration temperatures below 45°F based on Palumbo (1997) and input from the National Advisory Committee on Microbiological Criteria for Food (NACMCF 1999).

Temperature at each step (τ_{S2}, τ_{S3}, τ_{S4}) is modeled as a cumulative distribution in the following form:

TABLE 3-16 Storage Temperatures above 45°F

	Step 2 Retail/Wholesale Storage	Step 3 Transport	Step 4 Home/HRI Storage
Total samples	975	971	939
Temperature (°F)	Number of Samples above 45°F		
46	49	175	47
49	49	223	28
52	8	68	4
55	4	49	5
58	2	19	4
61	0	19	1
64	0	3	0
67	0	2	1
70	0	1	1

Source: Audits International 1999.

$$\tau_{SX} = \text{cumulative [(temperature), } (p)] \tag{3.31}$$

The cumulative distribution in Equation 3.31 consists of two arrays: the first array represents various temperatures shown in Table 3-16, and the second array represents the corresponding cumulative probability of each of the temperatures. In addition to modeling the variability in storage temperature as a cumulative distribution, uncertainty regarding the actual frequency of each temperature is modeled using a beta distribution after a method reported by Vose (1999).

Modeling Storage Time

The amount of time at each step (T_2, T_3, T_4) that ground beef is stored at a given temperature determines how much growth of *E. coli* O157:H7 takes place. Although there are recommendations for how long ground beef may be stored at temperatures above 45°F (FDA 1997), there are no data documenting this length of time. FSIS recommends that ground beef be stored in the refrigerator for no more than 2 days (FSIS 2000). For Steps 2 and 4, time of storage is modeled as an exponential distribution with a mean of 1. An exponential distribution was chosen because it has a single parameter and its probability density function is monotonically decreasing. In other words, using this function assumes that on average, ground beef is more likely to be stored for shorter times than for longer times. An exponential distribution with a mean of 1 predicts that 99% of ground beef will be stored less than 4.6 days. Additionally, uncertainty about the mean of the exponential distribution is modeled using a uniform distribution from 0.5 days to 1.5 days. An exponential distribution with a mean of 0.5 predicts that 99% of ground beef will be stored less than 2.3 days, while an exponential distribution with a mean of 1.5 predicts that 99% of ground beef will be stored less than 6.9 days. Equation 3.32 shows how time is modeled across the various uncertainties for Steps 2 and 4.

$$T_X = \text{exponential [uniform(0.5, 1.5)]} \tag{3.32}$$

For Step 3, the time of storage for transportation is based on data from Audits International (1999) (Table 3-17).

TABLE 3-17 Time of Transport from Retail to Home

Time of Transport	Number of Observations
0.00	36
0.25	5
0.50	46
0.75	168
1.00	240
1.25	210
1.50	156
1.75	67
2.00	28
2.25	10
2.50	8
2.75	3
3.00	1
6.50	1

Source: Audits International 1999.

In Step 3, time (T_3) is modeled as a cumulative distribution in the following form:

$$T_3 = \text{cumulative}[(\text{time}), (\text{p})] \quad (3.33)$$

As with Equation 3.31, the cumulative distribution in Equation 3.33 consists of two arrays. The first array represents the times shown in Table 3-17, and the second array represents the corresponding cumulative probability of each of the times. Again, uncertainty regarding the actual frequency of each time is modeled using a beta distribution after a method reported by Vose (1999).

Modeling the Effect of Freezing

Some ground beef may be frozen during storage and transportation. A decline in *E. coli* O157:H7 levels between 0 and 3 logs per gram of frozen ground beef is modeled based on laboratory studies of the effects of freezing on *E. coli* O157:H7 levels in ground beef (Ansay et al. 1999; Sage and Ingham 1998). Table 3-18 shows the frequency distribution used to model the log reduction of *E. coli* O157:H7 due to freezing. Uncertainty regarding the proportion of ground beef that is frozen is modeled uniformly from 20% to 80%.

Modeling Growth for a Single Step

Step 2 provides the first opportunity for *E. coli* O157:H7 growth. First, ln(LPD) is calculated given τ_{S2} using Equation 3.27. The lag period for Step 2 (LPD_2) is compared with the amount of time in Step 2 (T_2). If $LPD_2 < T_2$, then no growth occurs and the cumulative lag used in Step 2 (CLU_2) is as follows:

TABLE 3-18 Frequency Distribution for Log Reduction in *E. coli* O157:H7 due to Freezing

Log Reduction	Frequency
0.0	0.000
0.5	0.000
1.0	0.190
1.5	0.580
2.0	0.170
2.5	0.028
3.0	0.028

$$CLU_2 = \frac{LPD_2}{T_2} \tag{3.34}$$

If $LPD_2 > T_2$, then the amount of time available for growth equals $LPD_2 - T_2$. Equation 3.28 calculates $\ln(GT_2)$ given τ_{S2}. The log of growth for Step 2 (G_2) is then calculated as follows:

$$G_2 = \log_{10}\left(e^{\frac{LPD_2 - T_2}{GT_2}}\right) \tag{3.35}$$

CLU_2 or G_2 are only calculated if τ_{S2} is greater than 45°F. Otherwise both CLU_2 and G_2 are set at 0.

Since the CLU is modeled as a percentage that can increase across each step, the amount of *E. coli* O157:H7 growth in Steps 3 and 4 is also dependent on the CLU. If CLU_2 is greater than 0, then the LPD in Step 3 must be adjusted to account for the CLU. The adjusted LPD_3 (LPD_{3a}) is calculated by $LPD_3 \times (1 - CLU_2)$. Equations 3.34 and 3.35 can then be used to calculate G_3 by substituting LPD_{3a} where LPD_3 would occur. The amount of *E. coli* O157:H7 growth in Step 4 would be calculated in the same manner.

Modeling Growth across Steps 2 to 4

Since CLU and G are only modeled when storage temperature exceeds 45°F, there is a set of eight potential growth combinations that can occur in Steps 2 through 4 for a single ground beef serving. If the temperature of the ground beef serving is below 45°F, then growth is not modeled. If the serving is exposed to temperatures above 45°F in one of the three steps, then CLU and/or G is calculated for that step. If the temperature of the serving is above 45°F in two of the steps, then CLU and/or G is calculated for that step and an adjusted LPD is calculated if CLU is less than 1. The same principle applies if the temperature of the uncooked ground beef serving is above 45°F in all three steps. Thus, the total number of combinations of steps above or below 45°F is 2^3 or 8. The probability that a serving will be exposed to a particular combination of steps above 45°F is dependent on the probability of each step being above 45°F. These probabilities are considered fixed but uncertain.

The probability that a serving in a particular step will be above 45°F is modeled using a beta(s+1,n–s+1) distribution incorporating the data in Table 3-16, where s equals the total samples above 45°F and n equals the total samples. Consequently, a single simulation of the model will generate eight different growth distributions for each of the eight different combinations of steps above 45°F. The eight growth distributions generated from these eight

combinations are integrated across the probabilities of their occurrence to create an overall growth distribution for *E. coli* O157:H7 in stored ground beef. This distribution is then integrated with the distribution for freezing of ground beef to give a final distribution (G_{pop}) representing the change of *E. coli* O157:H7 due to storage in Steps 2 to 4 for all servings.

Figure 3-23 shows the results of 20 Monte Carlo simulations where G_{pop} is estimated. Each line represents the frequency distribution returned by a single simulation.

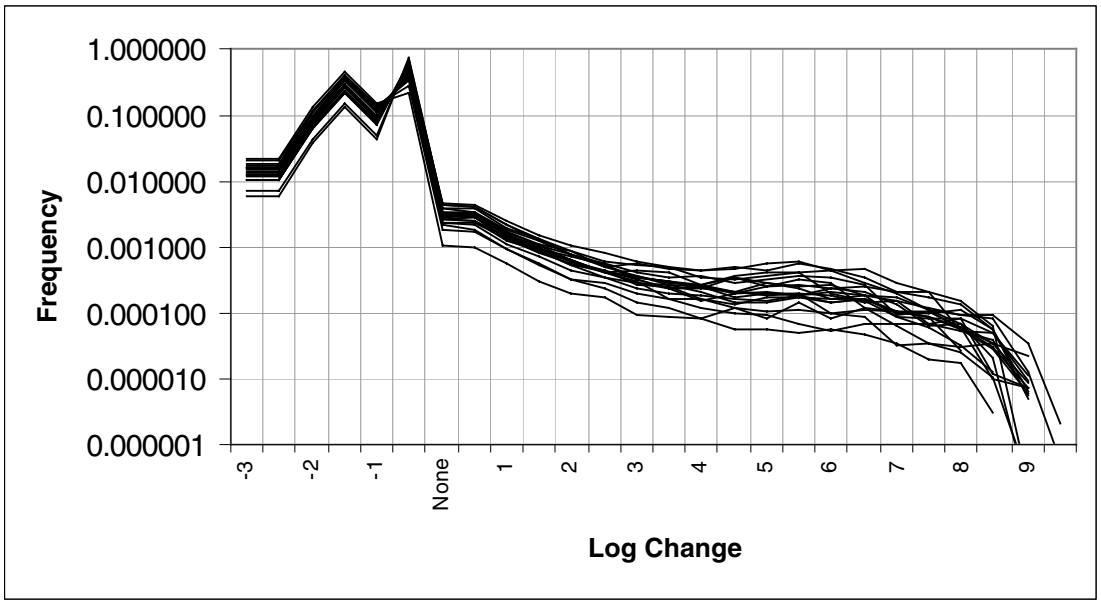

FIGURE 3-23 Frequency of log increase or log decrease due to storage for 20 simulations.

Cooking (Step 5)

Step 5 simulates the effect of cooking on ground beef in homes and HRI. Nearly all ground beef is cooked. The effect of cooking is dependent on the cooking temperature, the storage temperature prior to cooking, and the thermodynamics of the product. The effects of cooking temperature and precooking storage are modeled. Cooking is modeled by relating log reduction to internal product temperatures.

Temperatures of Cooked Ground Beef

The temperature to which a ground beef serving is cooked is based on a survey of final internal product temperatures of cooked hamburger patties prior to consumption (Audits International 1999). Table 3-19 shows the internal hamburger temperatures reported. Because visual cues are unreliable indicators of cooking of ground beef (Liu and Berry 1996; Van Laack et al. 1996), quantitative time-temperature cooking data were used (Audits International 1999) rather than consumer behavior survey data (Brent 1999).

TABLE 3-19 Internal Temperatures of Cooked Hamburger Patties

Internal Temperature (°C)	Observations (n)	Internal Temperature (°C)	Observations (n)
39	2	69	22
41	5	71	18
43	3	73	55
45	9	75	45
47	5	77	59
49	14	79	19
51	8	81	18
53	13	83	74
55	23	85	11
57	12	87	5
59	20	89	9
61	31	91	1
63	41	93	3
65	25	95	3
67	41	Total	594

Source: Audits International 1999.

Effect of Cooking

Juneja et al. (1997) determined the effect of cooking on hamburgers experimentally inoculated with an initial load of 6.6 logs of *E. coli* O157:H7. Final internal temperatures of the hamburgers ranged from 56°C to 74°C (133°F to 166°F). The log of the surviving *E. coli* O157:H7 was then measured. Given a temperature in Fahrenheit (τf), the following linear regression equation gives the corresponding ($r^2 = 0.94$) log reduction:

$$LR = 6.6 - (20.53 - 0.12 \times \tau f) \quad (3.36)$$

Juneja et al. (1997) noted that 73% lean ground beef patties (100 grams) cooked to an internal temperature of 68.3°C (155°F) would have a 4 log reduction of a five strain cocktail of *E. coli* O157:H7. This is consistent with a report by Jackson et al. (1996) that 78% lean ground beef patties (114 grams) inoculated with about 6 logs of bacteria and cooked to an internal temperature of 68.3°C (155°F) would have a 4.1 log reduction with a standard deviation of 0.5 logs. In both studies, inoculated hamburgers were stored under refrigeration.

Semanchek and Golden (1998) reported variability in heat resistance among three strains of *E. coli* O157:H7 and concluded that "exposure to different environments may select for resistance to suboptimum conditions or subsequent stress" (p. 399). Jackson et al. (1996) reported that the response of *E. coli* O157:H7 to cooking appeared to be related to original storage temperatures. *E. coli* O157:H7 in frozen ground beef was more heat resistant than *E. coli* O157:H7 in ground beef refrigerated or stored at higher temperatures. Jackson et al. reported

results from 27 different combinations of storage conditions and cooking temperatures (listed in Table 3-20).

TABLE 3-20 Mean Log Reductions (± Sample Standard Deviation [std. dev.]) of *E. coli* O157:H7 in Grilled Ground Beef Patties

Pretreatment Storage Conditions	Internal Cooking Temperature					
	54.4°C		62.8°C		68.3°C	
	Mean LR	Std. Dev.	Mean LR	Std. Dev	Mean LR	Std. Dev
–18°C, 8 days	0.3	0.1	1.2	0.6	3.0	1.8
–18°C, 8 days followed by 21°C, 4 hours	0.7	0.1	3.9	0.9	4.8	0.3
–18°C, 8 days followed by 30°C, 4 hours	1.6	0.7	5.5	0.3	5.2	0.7
3°C, 9 hours	0.5	0.3	2.6	0.5	4.1	0.5
3°C, 9 hours followed by 21°C, 4 hours	1.3	0.4	5.3	0.2	5.2	0.3
3°C, 9 hours followed by 30°C, 4 hours	1.9	0.3	6.0	0.2	5.8	0.4
15°C, 9 hours	1.0	0.1	4.3	0.7	5.1	0.1
15°C, 9 hours followed by 21°C, 4 hours	1.6	0.8	5.4	0.5	5.6	0.4
15°C, 9 hours followed by 30°C, 4 hours	2.4	0.1	5.3	1.7	6.4	0.2

Source: Jackson et al. 1996.

Modeling Cooking

The effect of cooking is calculated in the model by applying log reductions for the range of cooking temperatures shown in Table 3-19. Although Jackson et al. do not report on the effect of cooking at temperatures greater than 68.3°C (155°F), this effect was extrapolated in accordance with the linear relationship demonstrated by Juneja et al. (1997).

Figure 3-24 depicts the variability expected for log reductions across the nine different pretreatments shown in Table 3-20. Individual lines are not labeled, as the purpose of the chart is to show the wide range of variability in log reduction based solely on precooking storage.

The information in Table 3-20 is used to calculate a linear regression equation for each of the nine pretreatments with estimated y intercept (α), slope (β), and the standard error of y (stey) terms. For each regression equation, the probability of a particular log reduction for each of the 30 temperatures (τ_i) in Table 3-19 is calculated using the Excel Normdist function:

$$p(\mathrm{LR} \mid \tau_i) = \mathrm{NORMDIST}(\mathrm{LR}, \alpha + \beta \times \tau_i, \mathrm{stey}, 1) \quad (3.37)$$

Integrating the probabilities of all of the temperatures and the probability of a given log reduction across all τ_i results in a log reduction curve for a given pretreatment:

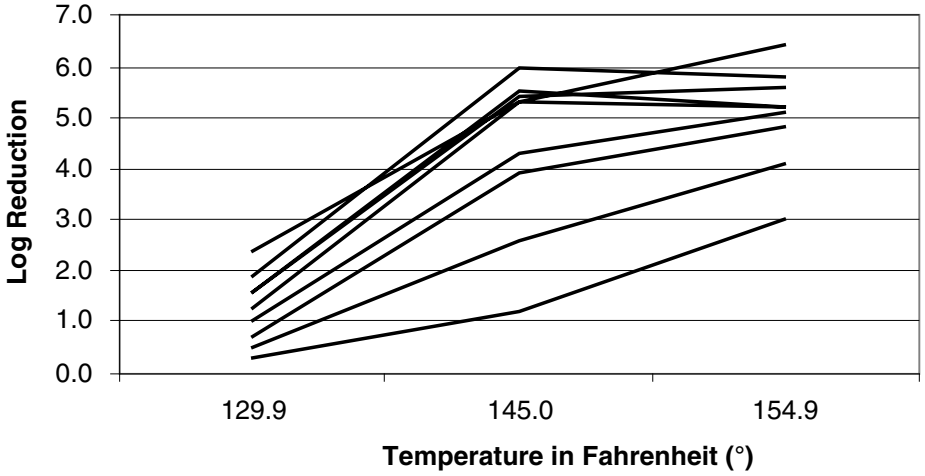

FIGURE 3-24 Variability in log reduction of *E. coli* O157:H7 for nine different pretreatments based on Jackson et al. (1996).

$$f(\mathrm{LR}) = \int_{39}^{95} (p(\mathrm{LR} \mid \tau) \times \propto p(\tau) d\tau \tag{3.38}$$

The probability of a particular pretreatment occurring for a ground beef serving is fixed but uncertain. These probabilities are dependent on probabilities used in Steps 2 to 4. For instance, the probability of the serving having undergone freezing before cooking is dependent on, and correlated with, the probability that the serving was frozen during Steps 2 to 4.

The log reduction curves for each of the nine pretreatments are integrated to create a single log reduction curve. This log reduction curve (LR_{pop}) describes the frequency of log reductions from cooking for the entire population of servings and is estimated using Monte Carlo methods.

Figure 3-25 shows 20 different LR_{pop} curves calculated from Monte Carlo simulations.

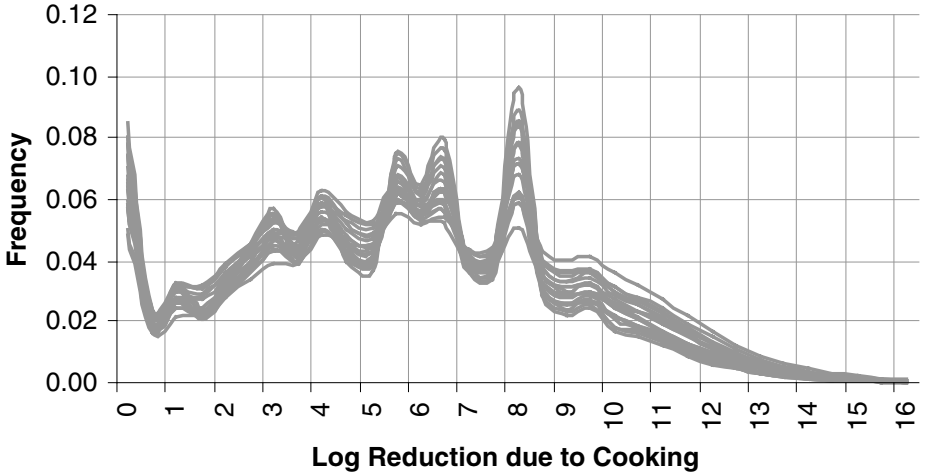

FIGURE 3-25 Frequency of log reduction due to cooking for 20 simulations.

Note that considerable uncertainty exists regarding the log reduction due to cooking. Note also that this particular set of simulations suggests that between 4% and 8% of servings have no log reduction applied.[2]

Food items with ground beef as a major ingredient were assumed to have cooking practices that parallel cooking practices for hamburgers. As noted in the scope, cooking of ground beef as an ingredient in products such as chili, spaghetti, and soup is assumed to destroy all *E. coli* O157:H7 in the product. Such ground beef is usually precooked in a granular form and then subjected to further cooking.

Although D'Sa et al. (2000) have reported a difference in log reductions between single-sided and double-sided cooking, this distinction was not modeled. Results from Juneja et al. (1997), Jackson et al. (1996), and D'Sa et al. (2000) were based on cooking similar sized hamburger patties of relatively uniform thickness. Consequently, this model did not explicitly account for differences in patty thickness. Nevertheless, the variability of internal cooking temperatures included in this model should account for the thermodynamics in ground beef servings with varying thickness.

Consumption (Step 6)

Types of Ground Beef Products Modeled

Ground beef is consumed in the United States as the main course of a meal or as an ingredient in a recipe both at home and away from home in HRI. Data from the 1994–1996, 1998 CSFII were used to model ground beef consumption patterns by age of the consumer and location where the meal was consumed. The CSFII is a national survey of U.S. food intakes that consists of the following:

- a nationally representative sample of 21,154 respondents;
- two 24-hour recalls of foods eaten during two nonconsecutive days (with the interview for the second day conducted on a different day of the week, 3 to 10 days after the interview for the first day);
- demographic information on consumers;
- location where the meal was consumed (i.e., home versus away from home); and
- annual and 4-year survey weights to reflect the consumption patterns of the noninstitutional U.S. population.

Three categories of ground beef meals were considered in this step: (1) raw ground beef, (2) hamburger patties and sandwiches, and (3) formed ground beef products in which the ground beef is a major ingredient to the product (e.g., meatballs and meat loaf). Food items for each category were selected from over 7,200 food items within the 1994–1996 and 1998 CSFII (Kause 2001). Tables 3-21, 3-22, and 3-23 provide detailed information on the food items that comprise each ground beef category. Only food items with at least one eating occasion between 1994 and 1996 or in 1998 were included.

[2]The 1994–1996, 1998 CSFII included four individuals (three between 25 and 64 years of age and one less than 5 years of age) who were reported to have consumed "raw" ground beef. These reported ground beef servings comprised less than 0.07% of the estimated annual number of ground beef servings consumed in the United States (Tables 3-24, 3-25, and 3-26). For modeling purposes, these servings are considered to be a subset of those servings that have no log reduction in *E. coli* O157:H7 during cooking (e.g., grossly undercooked servings).

TABLE 3-21 1994–96, 1998 CSFII Food Codes for Raw Ground Beef Meals

Food Code	Food Item
21500000	Raw ground beef
27116400	Steak tartare (raw ground beef and egg)

Amounts of Ground Beef Products Consumed

The amount of ground beef in each food item was calculated using the CSFII recipe files (Tables 3-24, 3-25, and 3-26). This provides information on the amount of ground beef consumed during a meal (e.g., meatball and spaghetti dinner).

Consumption data for each ground beef category were stratified by general location of where the meal was eaten (i.e., either at home or away from home). This resulted in six combinations for ground beef consumption by location: (1) raw ground beef consumed within the home, (2) raw ground beef consumed away from the home in HRI, (3) ground beef consumed as hamburgers within the home, (4) ground beef consumed as hamburgers away from the home in HRI, (5) ground beef used as a primary ingredient in a recipe (e.g., meatballs or meat loaf) within homes, and (6) ground beef used as an ingredient in a recipe (e.g., meatballs or meat loaf) away from the home.

Ground beef consumption was further stratified into four age categories (0 to 5, 6 to 24, 25 to 64, and 65+ years of age)[3] to provide more detail on exposure of susceptible age groups (0 to 5 and 65+ years of age). The age-specific annual number of ground beef meals consumed and the corresponding serving size (in grams) was calculated using SAS (version 8.0) and WesVar (version 2.0) software (Kause 2001). The following information was derived:

- weighted descriptive statistics (e.g., mean amount eaten in grams, number of eating occasions, and mean number of eating occasions) that characterize all age/location/food category-specific eating occasions consumed in two nonconsecutive days of eating;
- distributions of the amount of food (in grams) that is eaten at all eating occasions, expressed as weighted percentiles after adjustment for the stratified sample design using a jackknife procedure in the WesVarPC software package with replicate weights that accompany the 1994–96, 1998 CSFII data;
- weighted descriptive statistics to describe the amount of food (in grams) that is eaten per person per day and the number of consumers; and
- per capita estimates of food eaten.

The resulting number of servings and mean serving size of ground beef by age and location for each ground beef category are shown in Tables 3-24, 3-25, and 3-26. These ground beef meals account for over 18 billion ground beef servings consumed annually in the United States.

[3]Age categories were used instead of age-specific data because of the limited number of observations for each age (e.g., 1-year-olds, 2-year-olds, etc.) to derive the statistics.

TABLE 3-22 1994–96, 1998 CSFII Food Codes for Hamburger Patty and Sandwich Meals

Food Code	Food Item	Food Code	Food Item
21500100	Ground beef or patty	27510390	Double bacon cheeseburger, on bun
21500200	Ground beef or patty, breaded, cooked	27510400	Bacon cheeseburger, ¼ lb meat, with tomato, on bun
21501000	Ground beef, regular, cooked	27510420	Taco burger, on bun (include chiliburger with cheese)
21501200	Ground beef, lean, cooked	27510430	Double bacon cheeseburger, with mayo, tomato, on bun
21501300	Ground beef, extra lean, cooked	27510440	Bacon cheeseburger, ¼ lb, with mayo and tomato, on bun
25220140	Beef sausage, fresh, bulk, patty or link, cooked	27510480	Cheeseburger, with onions, on rye bun
27510210	Cheeseburger, plain, on bun	27510500	Hamburger, plain, on bun
27510220	Cheeseburger, with mayo, on bun	27510510	Hamburger, with tomato and or catsup, on bun
27510230	Cheeseburger, with mayo and tomato, on bun	27510520	Hamburger, with mayo and tomato, on bun
27510240	Cheeseburger, ¼ lb meat, plain, on bun	27510530	Hamburger, ¼ lb meat, plain, on bun
27510250	Cheeseburger, ¼ lb meat, with mayo, on bun	27510540	Double hamburger with tomato and or catsup, on bun
27510260	Cheeseburger, ¼ lb meat, with mushroom sauce, on bun	27510550	Double hamburger with mayo and tomato, double-decker bun
27510270	Double cheeseburger, plain, on bun	27510560	Hamburger, ¼ lb meat with mayo and tomato, on bun
27510280	Double cheeseburger, with mayo, on bun	27510590	Hamburger, with mayo, on bun
27510300	Double cheeseburger, with mayo, on double-decker bun	27510600	Hamburger, 1 oz meat, plain, on miniature bun
27510310	Cheeseburger, with tomato and or catsup, on bun	27510610	Hamburger, 1 oz meat, tomato, on miniature bun
27510311	Cheeseburger, 1 oz meat, plain, on mini bun	27510620	Hamburger, ¼ lb meat, with tomato and or catsup, bun
27510320	Cheeseburger, ¼ lb meat, with tomato/catsup, bun	27510630	Hamburger, ¼ lb meat, with mayo, on bun
27510330	Double cheeseburger, with tomato and or catsup, on bun	27510640	Hamburger, ¼ lb meat (modified fat) with tomato, on bun
27510340	Double cheeseburger, with mayo and tomato on bun	27510670	Double hamburger, with mayo and tomato, on bun
27510350	Cheeseburger, ¼ lb meat, with mayo and tomato on bun	27510680	Double hamburger (1/2 lb meat), with tomato/catsup, bun
27510360	Cheeseburger, with mayo, tomato and bacon on bun	27510690	Double hamburger, 1/2 lb meat, with mayo and tomato/catsup, bun
27510370	Double cheeseburger with mayonnaise, on bun	27510700	Meatball and spaghetti sauce submarine sandwich
27510380	Triple cheeseburger with mayo, tomato, on bun		

TABLE 3-23 1994–96, 1998 CSFII Food Codes for Other Ground Beef-Based Meals

Food Codes	Food Item
21500110	Ground beef, meatballs, meat only, not specified as to regular/lean
21540100	Ground beef with textured vegetable protein, cooked
23220010	Veal, ground or patty, cooked
27116350	Stewed, seasoned ground beef, Mexican style
27118110	Meatballs, p.r. (albondigas)
27118120	Stewed, seasoned ground beef, Puerto Rican style
27160100	Meatballs, not specified as to type of meat, with sauce
27161010	Meat loaf, p.r. (albondigon)
27214100	Meat loaf made with beef
27214110	Meat loaf with beef, with tomato sauce
27260010	Meat loaf, not specified as to type of meat
27260050	Meatballs, with breading, with gravy
27260080	Meat loaf made with beef and pork
27260090	Meat loaf with beef, veal and pork
27260100	Meat loaf with beef and pork, with tomato sauce
27113300	Swedish meatballs with cream or white sauce (mixture)

TABLE 3-24 Annual Number of Servings of Raw Ground Beef Consumed at Home and Away from Home by Age Category in the 1994–1996, 1998 CSFII

Age in Years	Number of Servings	Mean Serving Size (grams)
Home		
0–5	—	—
6–24	—	—
25–64	8,861,470	113.40
65+	—	—
Total	8,861,470	
Away from Home		
0–5	522,315	56.70
6–24	—	—
25–64	3,883,053	12.60
65+	—	—
Total	4,405,368	

TABLE 3-25 Annual Number of Servings of Hamburger Patties and Sandwiches Consumed at Home and Away from Home by Age Category in the 1994–1996, 1998 CSFII

Age in Years	Number of Servings	Mean Serving Size (grams)
Home		
0–5	395,592,840	51.86
6–24	1,478,341,250	95.17
25–64	2,517,532,750	102.02
65+	577,825,295	86.52
Total	4,969,292,135	
Away from Home		
0–5	717,308,950	36.88
6–24	4,215,244,840	78.73
25–64	5,628,291,058	87.64
65+	523,589,763	67.53
Total	11,084,434,611	

TABLE 3-26 Annual Number of Servings of Ground Beef-Based Meals (Such as Meat loaf and Meatballs) Consumed at Home and Away from Home by Age Category in the 1994–1996, 1998 CSFII

Age in Years	Number of Servings	Mean Serving Size (grams)
Home		
0–5	109,001,410	62.36
6–24	362,621,113	123.02
25–64	686,647,125	123.95
65+	272,269,925	100.09
Total	1,430,539,573	
Away from Home		
0–5	27,548,375	64.01
6–24	169,672,623	75.64
25–64	398,076,300	101.57
65+	135,376,128	67.30
Total	730,673,425	

Determining Exposures to E. coli O157:H7

The amount of *E. coli* O157:H7 to which a consumer is exposed in a single serving of ground beef is a function of the original number of *E. coli* O157:H7 organisms and the subsequent effects of storage, handling, and cooking on the growth or decline in the number of *E. coli* O157:H7 organisms in ground beef. The effect of storage on the growth or decline of organisms has been determined in Steps 2 to 4, and the effect of cooking has been determined in Step 5. The original number of organisms in a product is determined by the original concentration after grinding (Step 1) and the amount of product consumed (Step 6).

Equation 3.39 calculates the number of *E. coli* O157:H7 in a grinder load. The concentration in the grinder load (GLC) is calculated by dividing the total number of *E. coli* O157:H7 organisms (ECO) by the weight of the grinder load in grams as shown in the following equation where NCB is the number of combo bins in the grinder load, 2,000 is the weight of a combo bin in pounds, and 454 is the number of grams in a pound:

$$\mathrm{GLC} = \frac{\mathrm{ECO}}{\mathrm{NCB} \times 2{,}000 \times 454} \tag{3.39}$$

For a given GLC and a given serving size (WTG) the probability of having a particular number of organisms (BACT) in a serving is predicted by assuming a Poisson distribution

$$p(\mathrm{BACT}) = \frac{(\mathrm{GLC} \times \mathrm{WTG})^{\mathrm{BACT}}}{\mathrm{BACT!}} e^{-\mathrm{GLC} \times \mathrm{WTG}} \tag{3.40}$$

Integrating the probabilities of all GLCs and the probability of all BACTs across all WTGs results in an initial serving distribution:

$$f(\mathrm{BACT}) = \int_{\mathrm{GLC}=10^{-7}}^{10^{7}} \int_{\mathrm{WTG}=12}^{124} [\, p(\mathrm{BACT} \mid \mathrm{GLC}, \mathrm{WTG}) \times p(\mathrm{GLC}) \times p(\mathrm{WTG})\, d\mathrm{GLC}\, d\mathrm{WTG}] \tag{3.41}$$

This initial serving distribution describes the frequency of *E. coli* O157:H7 levels in all ground beef servings before storage and cooking ($\mathrm{BACT}_{\mathrm{pop}}$). This distribution is estimated for both the low prevalence and high prevalence seasons. Figure 3-26 shows the results of 20 Monte Carlo simulations where $\mathrm{BACT}_{\mathrm{pop}}$ is estimated for the low prevalence season and the high prevalence season.

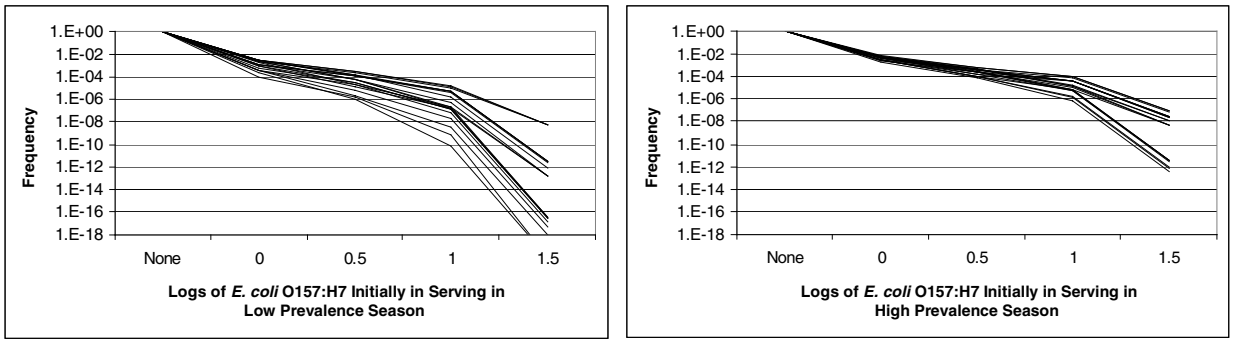

FIGURE 3-26 Frequency of logs of *E. coli* O157:H7 initially present in servings for low prevalence and high prevalence seasons.

The final dose distribution to which the population is exposed ($DOSE_{pop}$) can be expressed as the initial serving distribution plus the growth distribution minus the distribution describing the effect of cooking:

$$DOSE_{pop} = BACT_{pop} + G_{pop} - LR_{pop} \quad (3.42)$$

Recognize that $DOSE_{pop}$ is a distribution that represents the summation of three uncertain distributions. Although Monte Carlo methods could be used to combine these distributions, the model instead uses combinatorial mathematics to accomplish this.

Preparation Module Results

Grinder Loads

Grinder Loads Made from 2,000-Pound Combo Bins

An intermediate output of the preparation module is the distribution of *E. coli* O157:H7 densities in grinder loads of ground beef made from 2,000-pound combo bins. Figure 3-27 shows the results of 100 simulations for grinders in the low and high prevalence seasons. (See Appendix A for a discussion of how intermediate model outputs are anchored to observed ground beef sampling results.)

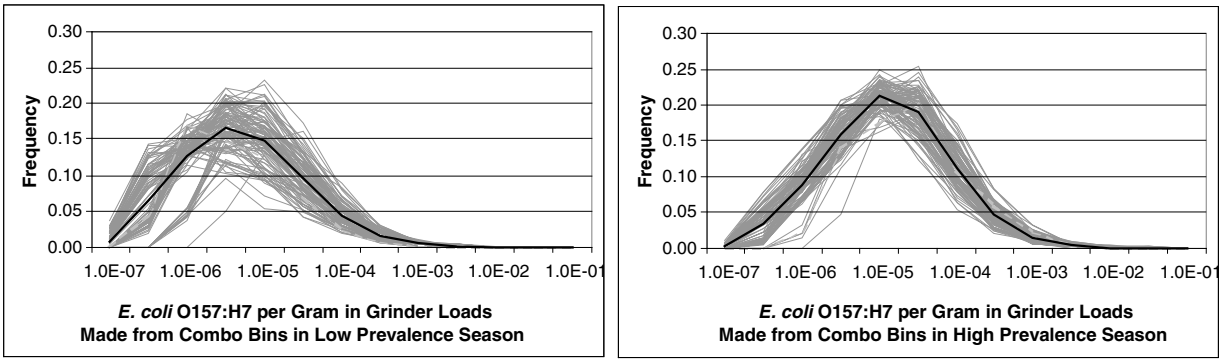

FIGURE 3-27 Frequency of ground beef contamination in contaminated grinder loads made from 2,000-pound combo bins in low and high prevalence seasons. Grinder loads that are not contaminated are not shown in this chart. The mean grinder load distribution is represented by the dark line.

Table 3-27 summarizes the prevalence of contaminated grinder loads for the 100 simulations depicted in Figure 3-27. The mean results imply that 32% of the grinder loads in the low prevalence season and 14% of the grinder loads in the high prevalence season are not contaminated.

In the low prevalence season, between 40% (5th percentile) and 88% (95th percentile) of these grinder loads contained one or more *E. coli* O157:H7. In the high prevalence season, between 61% (5th percentile) and 94% (95th percentile) of grinder loads contained one or more *E. coli* O157:H7.

TABLE 3-27 Results of 100 Simulations for Grinder Loads Constructed from 2,000-Pound Combo Bins in the Low and High Prevalence Seasons

	Percent Contaminated Grinder Loads	
	Low Prevalence Season	High Prevalence Season
Mean	68%	86%
Minimum	28%	61%
5th percentile	40%	76%
50th percentile	71%	88%
95th percentile	84%	93%
Maximum	88%	94%

Grinder Loads Made from 60-Pound Trim Boxes

Another intermediate output of the preparation module is the distribution of *E. coli* O157:H7 densities in grinder loads of ground beef made from 60-pound trim boxes. Figure 3-28 shows the results of 100 simulations for grinder loads constructed from these 60-pound boxes in the low and high prevalence seasons.

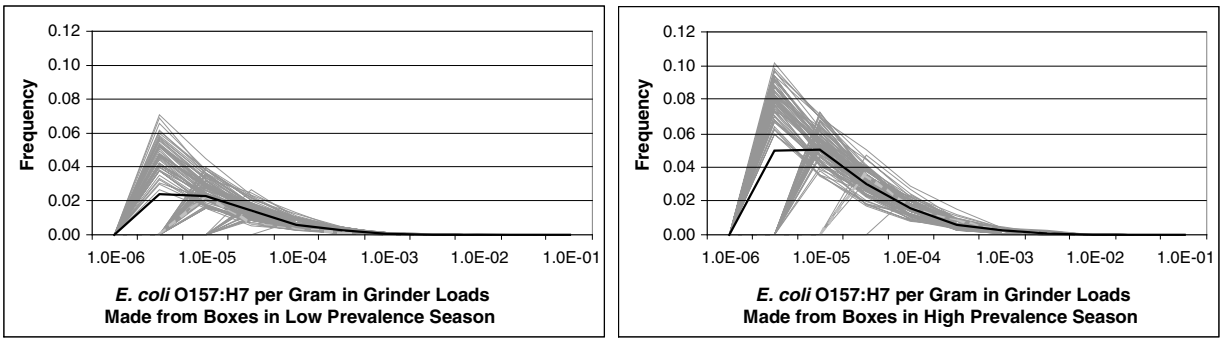

FIGURE 3-28 Frequency of ground beef contamination in contaminated grinder loads made from 60-pound trim boxes in low and high prevalence seasons. Grinder loads that are not contaminated are not shown in this chart. The mean grinder load distribution is represented by the dark line.

Table 3-28 summarizes the prevalence of contaminated grinder loads constructed from trim boxes for the 100 simulations depicted in Figure 3-29. The mean results imply that 84% of the grinder loads in the low prevalence season and 73% of the grinder loads in the high prevalence season are not contaminated.

In the low prevalence season, between 2% (5th percentile) and 13% (95th percentile) of grinder loads constructed from trim boxes contained one or more *E. coli* O157:H7. In the high prevalence season, between 7% (5th percentile) and 22% (95th percentile) of these grinder loads contained one or more *E. coli* O157:H7.

TABLE 3-28 Results of 100 Simulations for Grinder Loads Constructed from 60-Pound Trim Boxes in the Low and High Prevalence Seasons

	Percent Contaminated Grinder Loads	
	Low Prevalence Season	High Prevalence Season
Mean	7%	15%
Minimum	1%	4%
5th percentile	2%	7%
50th percentile	7%	16%
95th percentile	13%	22%
Maximum	16%	27%

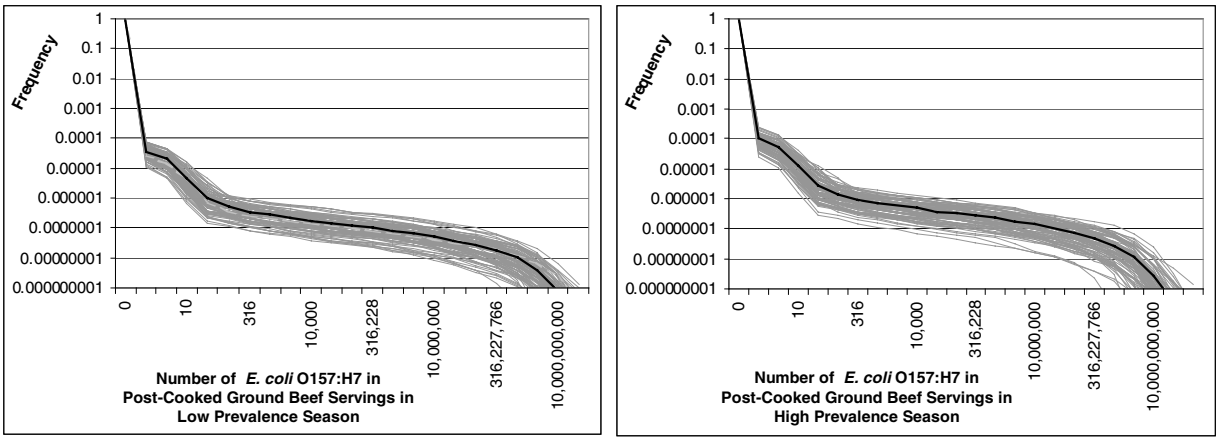

FIGURE 3-29 Frequency of exposure to various levels of *E. coli* O157:H7 during the low prevalence and high prevalence seasons. The mean exposure distribution for each is designated by the dark line.

Human Exposure to E. coli O157:H7

The primary outputs from the preparation module are distributions describing the prevalence of *E. coli* O157:H7 in ground beef servings generated during low and high prevalence seasons. These outputs become the inputs to risk characterization in which these exposure distributions are integrated with the output of hazard characterization to estimate risk of human illness from *E. coli* O157:H7 in ground beef. Figure 3-29 shows the results of 100 simulations for exposure distributions for the low and high prevalence seasons.

As shown in Figure 3-29, very few cooked ground beef servings are expected to have surviving *E. coli* O157:H7 organisms present. Table 3-29 summarizes the simulations shown in Figure 3-29. The mean results imply that 99.993% of cooked ground beef servings in the low prevalence season and 99.982% of cooked ground beef servings in the high prevalence season have no *E. coli* O157:H7 present. Furthermore, of the contaminated servings shown in Figure 3-29, about 95% have 10 or fewer *E. coli* O157:H7 organisms.

TABLE 3-29 Results of 100 Simulations Showing Percent of Post-Cooked Servings that Are Predicted to Have One or More Surviving *E. coli* O157:H7 in the Low and High Prevalence Seasons

	Percent Contaminated Servings	
	Low Prevalence Season	High Prevalence Season
Mean	0.007%	0.018%
Minimum	0.002%	0.004%
5th percentile	0.003%	0.007%
50th percentile	0.006%	0.019%
95th percentile	0.013%	0.030%
Maximum	0.014%	0.042%

Nevertheless, considerable uncertainty exists regarding the frequency of cooked ground beef servings that have 1 or more *E. coli* O157:H7 present. Table 3-29 implies that there is 90% confidence that the true frequency of contaminated servings lies somewhere between 1 in 36,000 and 1 in 7,600 in the low prevalence season and between 1 in 15,000 and 1 in 3,300 in the high prevalence season. In other words, there is a two- to threefold increase in the probability of consuming a contaminated serving in the high prevalence season compared with the low prevalence season. Such a difference mirrors the difference noted in FSIS ground beef sampling data between the high and low prevalence seasons (see Appendix A).

REFERENCES

American Meat Institute Foundation (AMIF). 1996, December. Relative Ground Beef Contribution to the United States Beef Supply. Final Report. Washington, DC: American Meat Institute Foundation.

Ansay, S.E., K.A. Darling, and C.W. Kaspar. 1999. Survival of *Escherichia coli* O157:H7 in ground-beef patties during storage at 2, –2, 15 and then –2 degrees C, and –20 degrees C. J Food Prot 62(11):1243-1247.

Animal and Plant Health Inspection Service (APHIS:VS:CEAH). 1994, March. *Escherichia coli* O157:H7 Issues and Ramifications. Fort Collins, CO.

Animal and Plant Health Inspection Service/National Animal Health Monitoring System (APHIS-VS-NAHMS). 1996, May. Part 1: Reference of 1996 Dairy Management Practices.

Animal and Plant Health Inspection Service/National Animal Health Monitoring System (APHIS-VS-NAHMS). 1997, June. Part 1: Reference of 1997 Beef Cow-Calf Management Practices.

Animal and Plant Health Inspection Service/National Animal Health Monitoring System (APHIS-VS-NAHMS). 1998, May. *E. coli* O157:H7 and Salmonella-Status on U.S. Dairy Operations.

Audits International. 1999. U.S. Cold Temperature Evaluation. Unpublished data.

Bacon, R.T., J.N. Sofos, K.E. Belk, et al. 1999. Evaluation of Multiple-Hurdle Microbiological Interventions—To Include Pre-Evisceration Spray-Washing and Other Technologies—For Beef Carcass Decontamination. Final Report. Submitted to National Cattlemen's Beef Association, March 3, 1999.

Bacon, R.T., K.E. Belk, J.N. Sofos, et al. 2000. Incidence of *Escherichia coli* O157:H7 on Hide, Carcass and Beef Trimmings Samples Collected from United States Packing Plants. Presented at FSIS Public Meeting on *E. coli* O157:H7, February 29, 2000, Arlington, VA.

Bailey, C., and R. Cox. 1976. The chilling of beef carcasses. Institutional Refrigeration Transport 72:76-90.

Bell, R.G. 1997. Distribution and sources of microbial contamination on beef carcasses. J Appl Microbiol 82:292-300.

Besser, T.E., D.D. Hancock, L.C. Pritchett, E.M. McRae, D.H. Rice, and P.I. Tarr. 1997. Duration of detection of fecal excretion of *Escherichia coli* O157:H7 in cattle. J Infect Dis 175:726-729.

Biela, T., Texas American Food Service. 1998. Personal communication.

Brent, C.P. 1999. Analysis of the Menu Census Survey Supplement—Hamburger and Egg Preparation Diary and Hamburger Consumption Quiz—for Consumer Storage and Handling Behaviors associated with Ground Beef. Informational memorandum to Margaret Coleman through Betsey Kuhn, Director, Food and Rural Economics Division, Economic Research Service, Washington, DC.

Brewer, B., Food Safety and Inspection Service (Washington, DC). February 1999. Personal communication.

Buchanan, R., and L. Bagi. 1994. Expansion of response surface models for the growth of *E. coli* O157:H7 to include sodium nitrite as a variable. Int J Food Microbiol 23:317-332.

Buchanan, R., and L. Bagi. 1997. Microbial competition: Effect of culture conditions on the suppression of Listeria monocytogenes Scott A by Carnobacterium piscicola. J Food Prot 60:254-261.

Cassin, M.H., A.M. Lammerding, E.C. Todd, W. Ross, and R.S. McColl. 1998. Quantitative risk assessment of *Escherichia coli* O157:H7 in ground beef hamburgers. Int J Food Microbiol 41:21-44.

Charlebois, R., R. Trudel, and S. Messier. 1991. Surface contamination of beef carcasses by fecal coliforms. J Food Prot 54:950-956.

Cornell Collaborative Project. April 1998. Prevalence of *E. coli* O157:H7 Pre-Slaughter/*E. coli* O157:H7 Associated Risk Factors. New York State Cull Cow Sub-Project. Final Report.

Cray, W.C., and H.W. Moon. 1995. Experimental infection of calves and adult cattle with *E. coli* O157:H7. Appl Environ Microbiol 61:1586-1590.

Cray, W.C., T.A. Casey, B.T. Bosworth, and M.A. Rasmussen. 1998. Effect of dietary stress on fecal shedding of *E. coli* O157:H7 in calves. Appl Environ Microbiol 64:1975-1979.

D'Sa, E.M., M.A. Harrison, S.E. Williams, and M.H. Broccoli. 2000. Effectiveness of two cooking systems in destroying *Escherichia coli* O157:H7 and *Listeria monocytogenes* in ground beef patties. J Food Prot 63(7):894-899.

Dargatz, D.A., S.J. Wells, L.A. Thomas, D. Hancock, and L.P. Garber. 1997. Factors associated with the presence of *Escherichia coli* O157 in feces in feedlot cattle. J Food Prot 60:466-470.

Delazari, I., S.T. Iaria, H. Riemann, D.O. Cliver, and N. Jothikumar. 1998a. Removal of *Escherichia coli* O157:H7 from surface tissues of beef carcasses inoculated with wet and dry manure. J Food Prot 61(10):1265-1268.

Delazari, I., S.T. Iaria, H.P. Riemann, D.O. Cliver, and T. Mori. 1998b. Decontaminating beef for *Escherichia coli* O157:H7. J Food Prot 61:547-550.

Diez–Gonzalez, F., T.R. Callway, and M.G. Kizoulis. 1998. Grain feeding and the dissemination of acid resistant *E. coli* from cattle. Science 281:1666-1668.

Dorsa, W. 1997. Review: New and established carcass decontamination procedures commonly used in the beef-processing industry. J Food Prot 60:1146-1151.

Dorsa, W., C. Cutter, and G. Siragusa. 1997. Effects of acetic acid, lactic acid and trisodium phosphate on the microflora of refrigerated beef carcass surface tissue inoculated with *Escherichia coli* O157:H7, *Listeria Innocua*, and *Clostridium Sporogenes*. J Food Prot 60:619-624.

Duewer, L. 1998. Updated beef carcass-to-retail consumption conversion factor increases to 0.7. Livestock, Dairy and Poultry Situation and Outlook. LDP-M-51, ERS. Pages 11-12.

Duffes, F., F. Leroi, P. Boyaval, and X. Dousset. 1999. Inhibition of Listeria monocytogenes by Carnobacterium spp. strains in a simulated cold smoked fish system stored at 4°C. Int J Food Microbiol 47:33-42.

Elder, R.O., J.E. Keen, G.R. Siragusa, G.A. Barkocy-Gallagher, M. Koomaraie, and W.W. Laegreid. 2000. Correlation of enterohemorrhagic *E. coli* O157 prevalence in feces, hides, and carcasses of beef cattle during processing. Proc Natl Acad Sci 97(7):2999-3003.

Farrell, B.L., A.B. Ronner, and A.C. Wong. 1998. Attachment of *Escherichia coli* O157:H7 in ground beef to meat grinders and survival after sanitation with chlorine and peroxyacetic acid. J Food Prot 119:817-822.

Federal Register. July 7, 1998. Technologies for the detection and reduction of pathogens to improve food safety. 63(129):36654-36655.

Food and Drug Administration (FDA). 1997. Food Code, 1997—Recommendations of the United States Public Health Service. National Technical Information Service PB97-133656.

Food Safety and Inspection Service (FSIS). 1994. Nationwide Beef Microbiological Baseline Data Collection Program: Steers and Heifers—October 1992–September 1993.

Food Safety and Inspection Service (FSIS). 1996. Directive #6350.1. Washington, DC.

Food Safety and Inspection Service (FSIS). 1998. Animal Disposition Reporting System. (Available at: www.fsis.usda.gov/ophs/adrsdata/).

Food Safety and Inspection Service (FSIS). 2000. Focus On Ground Beef. Consumer Education and Information. (Available at: http://www.fsis.usda.gov/OA/pubs/focusgb.htm.)

Galland, J.C. 1997. Risks and prevention of contamination of beef carcasses during the slaughter process in the United States of America. Contamination of animal products: Prevention and risks for public health. Reveu Scientifique et Technique, Office International Des Epizooties.

Garber, L.P., S.J. Wells, D.D. Hancock, M.P. Doyle, J. Tuttle, J.A. Shere, and T. Zhao. 1995. Risk factors for fecal shedding of *Escherichia coli* O157:H7 in dairy calves. J Am Vet Med Assoc 207:46-49.

Garber, L., S. Wells, L. Schroeder–Tucker, and K. Ferris. 1999. Factors associated with the shedding of verotoxin-producing *Escherichia coli* O157 on dairy farms. J Food Prot 62(4):307-312.

Getz, D., LeFiell Company (Omaha, NE). January 1999. Personal communication.

Gill, C.O. 1979. Intrinsic bacteria in meat. J Appl Bacteriol 47:367-378.

Gill, C.O. 1996. Cold storage temperature fluctuations and predicting microbial growth. J Food Prot 43-47.

Gill, C.O. 1998. Apparatus for Pasteurizing Red Meat Carcasses. Agriculture and Agri-Food Canada, Research Branch Technical Bulletin 1998-5E.

Gill, C.O. 1999. HACCP by guesswork or by the numbers? Food Quality Jan/Feb:28-32.

Gill, C.O., and M. Badoni. 1997. The hygienic and organoleptic qualities of ground beef prepared from manufacturing beef pasteurized by immersion in hot water. Meat Sci 46:67-75.

Gill, C.O., and J. Bryant. 1997. Assessment of the hygienic performances of two beef carcass cooling processes from product temperature history data or enumeration of bacteria on carcass surfaces. Food Microbiol 14:593-602.

Gill, C.O., M. Badoni, and T. Jones. 1996a. Hygienic effects of trimming and washing operations in a beef-carcass-dressing process. J Food Prot 59:666-669.

Gill, C.O., J. McGinniss, and M. Badoni. 1996b. Assessment of the hygienic characteristics of a beef carcass dressing process. J Food Prot 59:136-140.

Gill, C.O., M. Badoni, and J. McGinnis. 1999. Assessment of the adequacy of cleaning of equipment used for breaking beef carcasses. Int J Food Microbiol 46:1-8.

Grau, F.H. 1987. Prevention of microbial contamination in the export beef abattoir. Pp. 221-233 in Elimination of Pathogenic Organisms from Meat and Poultry, F.J.M. Smulders, ed. Amsterdam: Elsevier Press.

Grau, F.H., and P.B. Vanderlinde. 1992. Occurrence, numbers, and growth of Listeria monocytogenes on some vacuum-packaged processed meats. J Food Prot 55:4-7.

Gyles, C. 1998. Vaccines and shiga-toxin-producing *Escherichia coli* in animals. Pp. 434-444 in *Escherichia coli* O157:H7 and Other Shiga Toxin-Producing *E. coli* Strains, J.B. Kaper and A.D. O'Brien, eds. Washington, DC: ASM Press.

Haas, C., J. Rose, and J. Gerba. 1999. Quantitative Microbial Risk Assessment. New York: John Wiley & Sons.

Hancock, D. 2001. Personal communication. Ongoing research project in collaboration with FDA-CVM.

Hancock, D.D., T.E. Besser, M.L. Kinsel, P.I. Tarr, D.H. Rice, and M.G. Paros. 1994. The prevalence of *Escherichia coli* O157 in dairy and beef cattle in Washington State. Epidemiol Infect 113:199-207.

Hancock, D.D., T.E. Besser, D.H. Rice, D.E. Herriott, and P.I. Tarr. 1997a. A longitudinal study of *Escherichia coli* O157 in fourteen cattle herds. Epidemiol Infect 118:193-195.

Hancock, D.D., D.H. Rice, D.E. Herriot, T.E. Besser, E.D. Ebel, and L.V. Carpenter. 1997b. Effects of farm manure handling practices on *Escherichia coli* O157 prevalence in cattle. J Food Prot 60:363-366.

Hancock, D.D., D.H. Rice, L.A. Thomas, D.A. Dargatz, and T.E. Besser. 1997c. Epidemiology of *Escherichia coli* O157 in feedlot cattle. J Food Prot 60:462-465.

Hancock, D., T. Besser, and D. Rice. 1998a. Ecology of *Escherichia coli* O157:H7 in cattle and import of management practices. Pp. 85-91 in *Escherichia coli* O157:H7 and Other Shiga Toxin-Producing *E. coli* Strains, J.B. Kaper and A.D. O'Brien, eds. Washington, DC: ASM Press.

Hancock, D.D., T.E. Besser, D.H. Rice, E.D. Ebel, D.E. Herriott, and L.V. Carpenter. 1998b. Multiple sources of *Escherichia coli* O157 in feedlots and dairy farms in the northwestern United States. Prev Vet Med 35:11-19.

Hancock, D.D., D.H. Rice, and T.E. Besser. 1999. Prevalence of *E. coli* O157:H7 in feedlot cattle at slaughter plants. Study funded by National Cattlemen's Association. Washington State University.

Hancock, D., T. Besser, J. Lejeune, M. Davis, and D. Rice. 2001. The control of VTEC in the animal reservoir. Int J Food Microbiol 66:71-78.

Hardin, M.G., L. Acuff, L.J. Oman, et al. 1995. Comparison of methods for contamination removal for beef carcass surfaces. J Food Prot 58:368-374.

Herriot, D.E., D.D. Hancock, E.D. Ebel, L.V. Carpenter, D.H. Rice, and T.E. Besser. 1998. Association of herd management factors with colonization of dairy cattle by shiga toxin-positive *E. coli* O157. J Food Prot 61:802-807.

Heuvelink, A.E., F.L. Van den Biggelaar, J. Zwartkruis–Nahuis, R.G. Herbes, R. Huyben, N. Nagelkerke, W.J. Melchers, L.A. Monnens, and E. de Boer. 1998. Occurrence of verocytotoxin-producing *Escherichia coli* O157 on Dutch dairy farms. J Clin Microbiol Dec:3480-3487.

Jackson, T.C., M.D. Hardin, and G.R. Acuff. 1996. Heat resistance of *Escherichia coli* O157:H7 in a nutrient medium and in ground beef patties as influenced by storage and holding temperatures. J Food Prot 59:230-237.

Jameson, J.F. 1962. A discussion of the dynamics of *Salmonella* enrichment. J Hygiene Cambridge, 60:193-207.

Jordan, D. 1998. Pre-slaughter control of beef carcass contamination with *Escherichia coli* O157:H7: A risk assessment approach. Ph.D. dissertation, University of Guelph.

Jordan, D., S. McEwen, A. Lammerding, W. McNab, and J. Wilson. 1999. Pre-slaughter control of *Escherichia coli* O157 in beef cattle: A simulation study. Prev Vet Med 41:55-74.

Juneja, V.K., O.P. Snyder Jr., A.C. Williams, and B.S. Marmer. 1997. Thermal destruction of *Escherichia coli* O157:H7 in hamburger. J Food Prot 60:1163-1166.

Kastner, C. 1998. Research Highlights. Kansas State University Food Safety Consortium Annual Report.

Kause, J. 2001. SAS Analysis of the 1994–1996, 1998 CSFII for Consumption of Ground Beef by Age and Location for the *E. coli* O157:H7 Risk Assessment. Informational memorandum to Wayne Schlosser through Carol Maczka, Director, Risk Assessment Branch, Office of Public Health and Science, Food Safety and Inspection Service, USDA, Washington, DC.

Labadie, J., P. Gouet, and J. Fourand. 1977. Blood poisonings at slaughter and their consequences. Zentralbl Bakteriol Parasitenkd Infectionskr Hyg Abt 1 Orig Reihe B 164:390-396.

Lagreid, W.W., R.O. Elder, and J.E. Keen. 1999. Prevalence of *E. coli* O157:H7 in range calves at weaning. Epidemiol Infect 123(2):291-298.

Liu, M.N., and B.W. Berry. 1996. Variability in color, cooking times, and internal temperature of beef patties under controlled cooking conditions. J Food Prot 59:969-975.

Marks, H.M., M.E. Coleman, C.T.J. Lin, and T. Roberts. 1998. Topics in risk assessment: Dynamic flow tree process. Risk Anal 18:309-328.

Martin, S.W., A.H. Meek, and P. Wilberg. 1987. Veterinary Epidemiology. Ames, Iowa: Iowa State University Press.

McAloon, T., Cargill. March 1999. Personal communication.

Mechie, S.C., P.A. Chapman, and C.A. Siddons. 1997. A fifteen month study of *E. coli* O157:H7 in a dairy herd. Epidemiol Infect 118(1):17-25.

National Advisory Committee on Microbiological Criteria for Foods (NACMCF). 1993. Report on generic HACCP for raw beef. Food Microbiol 10:449-488.

National Advisory Committee on Microbiological Criteria in Foods (NACMCF). 1997. Minutes from November 20, 1997, meeting.

National Advisory Committee on Microbiological Criteria in Foods (NACMCF). 1999. Minutes from December 10, 1999, meeting.

National Agricultural Statistics Service (NASS). 1998. Livestock Slaughter: 1997 Summary. MtAn1-2-1(98):5,15.

New Zealand Public Health Report. 2000, July/August. Shiga-Toxin Producing *Escherichia coli* (STEC) Cases Increase Again in 1999. ISSN 1173-0250. Vol. 8, No. 7/8.

Newton, K.G., J.C. Harrison, and A. Wauters. 1978. Sources of psychotropic bacteria on meat at the abattoir. J Appl Bacteriol 45(1):75-82.

Nilsson, L., L. Gram, and H.H. Huss. 1999. Growth control of *Listeria monocytogenes* on cold-smoked salmon using a competitive lactic acid bacteria flora. J Food Prot 62:336-342.

Nutsch, A.L., R.K. Phebus, M.J. Riemann, J.S. Kotrola, D.E. Schaefer, J.E. Boyer, Jr., R.C. Wilson, J.D. Leising, and C.L. Castner. 1997. Evaluation of a steam pasteurization process in a commercial beef processing facility. J Food Prot 60:485-492.

Nutsch, A.L., R.K. Phebus, M.J. Riemann, J.S. Kotrola, R.C. Wilson, J.E. Boyer, Jr., and T.L. Brown. 1998. Steam pasteurization of commercially slaughtered beef carcasses: Evaluation of bacterial populations at five anatomical locations. J Food Prot 61:571-577.

Phebus, R., Kansas State University (Manhattan, KS). 1999. Personal communication.

Phebus, R.K., A.L. Nutsch, D.E. Schafer, R.C. Wilson, M.J. Riemann, J.D. Leising, C.L. Kastner, J.R. Wolf, and R.K. Prasai. 1997. Comparison of steam pasteurization and other methods for reduction of pathogens on surfaces of freshly slaughtered beef. J Food Prot 60:476-484.

Prasai, R., R.K. Phebus, C.M. Garcia Zepeda, C.C. Kastner, A.E. Boyle, and D.Y.C. Fung. 1995. Effectiveness of trimming and/or washing on microbiological quality of beef carcass. J Food Prot 58:1114-1177.

Puyalto, C., C. Colmin, and A. Laval. 1997. *Salmonella typhimurium* contamination from farm to meat in adult cattle. Descriptive study. Vet Res 28:449-460.

Rice, D.H., E.D. Ebel, D.D. Hancock, D.E. Herriott, and L.V. Carpenter. 1997. *Escherichia coli* O157 in cull dairy cows on farm, and at slaughter. J Food Prot 60:1-2.

Robins-Browne, R.M., E. Elliot, and P. Desmarchelier. 1998. Shiga toxin-producing *Escherichia coli* in Australia. In *Escherichia coli* O157:H7 and Other Shiga Toxin-Producing *E. coli* Strains, J.B. Kaper and A.D. O'Brien, eds. Washington, DC: ASM Press.

Ross, T. and T.A. McMeekin. 1991. Predictive microbiology: Applications of a square root model. Food Australia 43:202-207.

Sage, J.R., and S.C. Ingham. 1998. Survival of *Escherichia coli* O157:H7 after freezing and thawing in ground beef patties. J Food Prot 61(9):1181-1183.

Sanderson, M.W., J.M. Gay, D.D. Hancock, C.C. Gray, L.K. Fox, and T.E. Besser. 1995. Sensitivity of bacteriologic culture for detection of *Escherichia coli* O157:H7 in bovine feces. J Clin Microbiol 33(10):2616-2629.

Sargeant, J.M., J.R. Gillespie, R.D. Oberst, R.K. Phebus, D.R. Hyatt, L.K. Bohra, and J.C. Galland. 2000. Results of a longitudinal study of the prevalence of *E. coli* O157:H7 on cow-calf farms. Am J Vet Res 61(11):1375-1379.

Scanga, J.A., A.D. Grona, K.E. Belk, J.N. Sofos, and G.R. Smith. 2000. Microbiological contamination of raw beef trimmings and ground beef. Meat Sci 56:145-152.

Semanchek, J.J., and D.A. Golden. 1998. Influence of growth temperature on inactivation and injury of *Escherichia coli* O157:H7 by heat, acid, and freezing. J Food Prot 61:395-401.

Sheridan, J., B. Lynch, and D. Harrington. 1992. The effect of boning plant cleaning on the contamination of beef cuts in commercial boning hall. Meat Sci 32:155-164.

Siragusa, G.R., W.J. Dorsa, C.N. Cutter, G.L. Bennett, J.E. Keen, and M. Koohmaraie. 1998. The incidence of *Escherichia coli* on beef carcasses and its association with aerobic mesophilic plate count categories during the slaughter process. J Food Prot 61:1269-1274.

Smeltzer, T., B. Peel, and G. Collins. 1998. The role of equipment that has direct contact with the carcass in the spread of *Salmonella* in beef abattoir. Aust Vet J 55:275-277.

Smith, D.R. 1999. Designing Strategies for Food Safety Pathogen Containment in Cattle Feedyards. Presented at Annual Meeting of U.S. Animal Health Association, San Diego, CA.

Smith, G., Colorado State University—Meat Sciences Department. 1998. Personal communication.

Spika, J.S., R. Khakhria, P. Michel, D. Milley, J. Wilson, and J. Waters. 1998. Shiga toxin-producing *Escherichia coli* infections in Canada. In *Escherichia coli* O157:H7 and Other Shiga Toxin-Producing *E. coli* Strains, J.B. Kaper and A.D. O'Brien, eds. Washington, DC: ASM Press.

Van Donkersgoed, J., K.W.F. Jericho, and H. Grogan. 1997. Preslaughter hide status of cattle and the microbiology of carcasses. J Food Prot 60(12):1502-1508.

Van Donkersgoed, J., T. Graham, and V. Gannon. 1999. The prevalence of vertoxins, *E. coli* O157:H7, and *Salmonella* in the feces and rumen of cattle at processing. Can Vet J 40:332-338.

Van Laack, R.L.J.M., W.B. Berry, and M.B. Solomon. 1996. Effect of precooking conditions on color of cooked beef patties. J Food Prot 59:976-983.

Vose, D.J. 1996. Quantitative Risk Analysis: A Guide to Monte Carlo Simulation Modeling. West Sussex, England: John Wiley and Sons, Ltd.

Vose, D.J. 1999. Fitting second order nonparametric distributions to data using Bayes' theorem, Unpublished manuscript.

Walls, I., and V.N. Scott. 1996. Validation of predictive mathematical models describing the growth of *Escherichia coli* O157:H7 in raw ground beef. J Food Prot 59:1331-1335.

Zhao, T., M.P. Doyle, J. Shere, and L. Garber. 1995. Prevalence of enterohemorrhagic *Escherichia coli* O157:H7 in a survey of dairy herds. Appl Environ Microbiol 61:1290-1293.